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REMOVABLE CROSSED WIRES FOR FRACTURES INTO JOINTS

WALTER G. STUCK, M. D.

San Antonio

THE treatment of fractures involving such joints as the knee, ankle, or elbow is often complicated by the presence of small fragments of bone which are displaced so that they cannot be replaced in their normal position without open operation. Moreover, after open reduction there is usually need for some sort of internal fixation to hold these small fragments where they belong. Similarly in old malunited fractures which involve the joints, it is generally necessary to explore the fracture, replace the fragments, and fix them with some sort of suture, bone pegs, screws, plates or wires. More often than not the small fragments in old fractures are so soft and osteoporotic that the use of any kind of screw or nail will crush them. Nevertheless to secure good function of joints, in which a fracture has involved the articular surface, and to forestall subsequent disabling traumatic arthritis, it is essential to reduce the fractures as perfectly as possible.

Scores of devices of all types have been recommended for fixation of fractures, especially since Lane's popularization of the metal bone plate a generation ago. Metal screws or wires or bands are frequently the only appliances with which these fractures can be maintained in position for proper healing. At best, though, all appliances are foreign bodies which cause some irritation of bone or interference with healing of the fractures. Subsequent operations are therefore needed to remove the fixation devices after they have served their purpose.

In this connection, Venable, Stuck and Beach[1] reported, in 1936, the results of their comprehensive experiments on the reaction of

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bone to metals. They discovered that metal appliances caused irritation and destruction of bone by electrolytic reaction between the component metals of the appliance. They found further that most

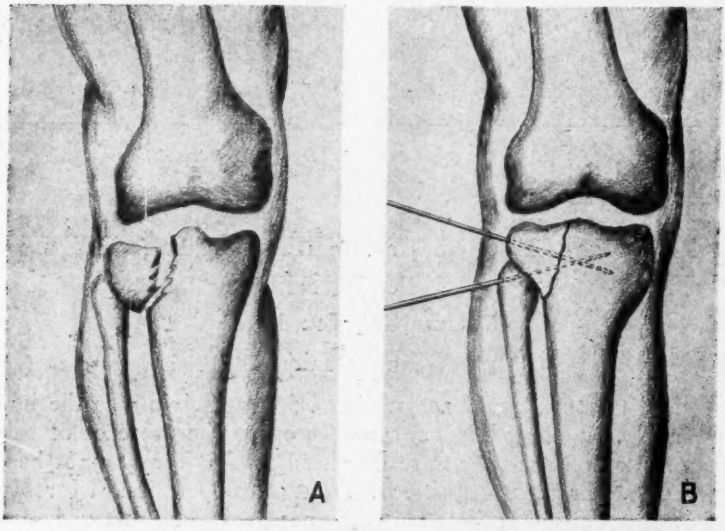

Fig. 1. *A*. Diagram showing fracture of external condyle of the tibia, with lateral displacement. *B*. Diagram of same fracture after reduction. Two Kirschner wires have been drilled through the fragment into the shaft of the bone, thus locking the fracture.

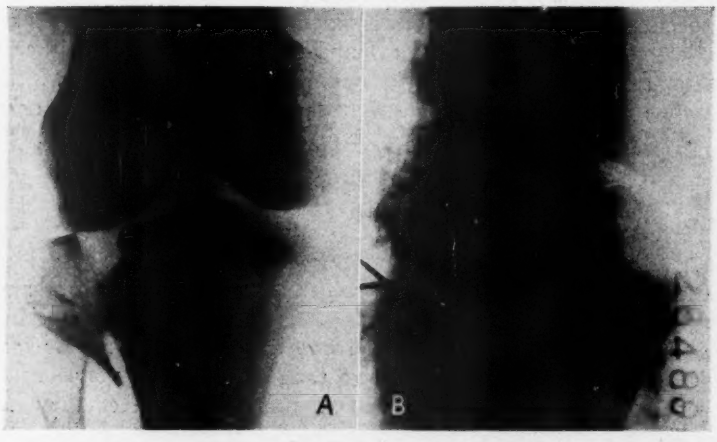

Fig. 2. M. G. Male, 37. *A*. Fracture of external condyle of the right tibia. Small fragment of bone in the fracture line. *B*. X-ray after reduction. Small scrap of bone removed from between fragments and fracture perfectly reduced. Locked in place with crossed Kirschner wires. Wires removed at the end of 4 weeks. Excellent function.

alloys used in bone surgery are composed of metals between which there is marked electrolytic reaction which destroys bone. Also metal devices plated with chromium, silver, copper, etc., caused similar reactions destructive to bone since the metal plating became cracked and thus allowed different metals to come in contact in the electrolyte (body fluids).

Because most metals will cause interference with bone growth, I have been using a simple method for fixing fragments of fractures by Kirschner wires so placed that they can be removed without subsequent operation. In this way fractures can be immobilized per-

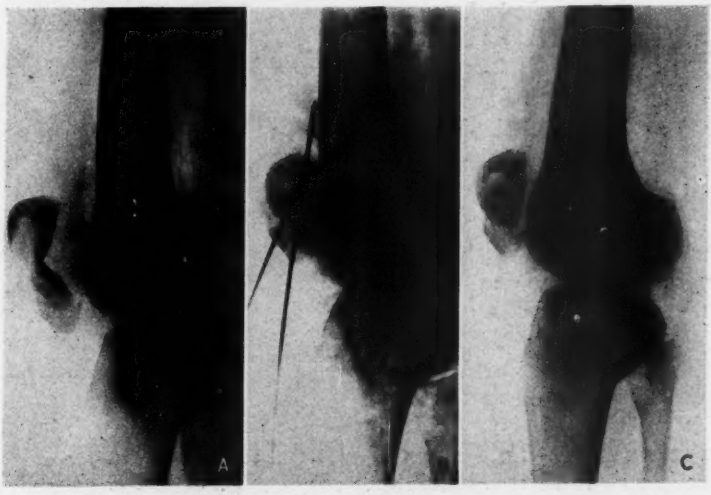

Fig. 3. I. J. Female, 25. *A*. Fracture of right patella in automobile accident with wide separation of fragments. *B*. Same fracture after reduction. Fragments held in place with two crossed Kirschner wires. Exercises begun at the end of three weeks with wires in place. Wires removed at the end of six weeks. *C*. Result two months after the accident. Solid bony union with good function of the joint.

fectly as long as necessary yet the wires can be removed easily as soon as they have served their purpose.

The fracture, whether recent or old, is explored and the fragments restored to their normal relation. Then two small Kirschner wires are drilled at a 15 to 30 degree angle to each other through the fragment and into the shaft of the bone. The fragments are thus securely locked together so that they cannot move in any direction. In this way there is a minimum amount of metal at the fracture line, the wires drill their own path and therefore cause no crushing of bone, and mechanically they are able to withstand the forces which tend to displace the fracture.

After the two wires are placed in the bone, the operative wound is closed and a cast applied to immobilize the extremity. The wires are left protruding through the skin and are protected by a pad of cotton covered with a few turns of plaster. At the end of four weeks

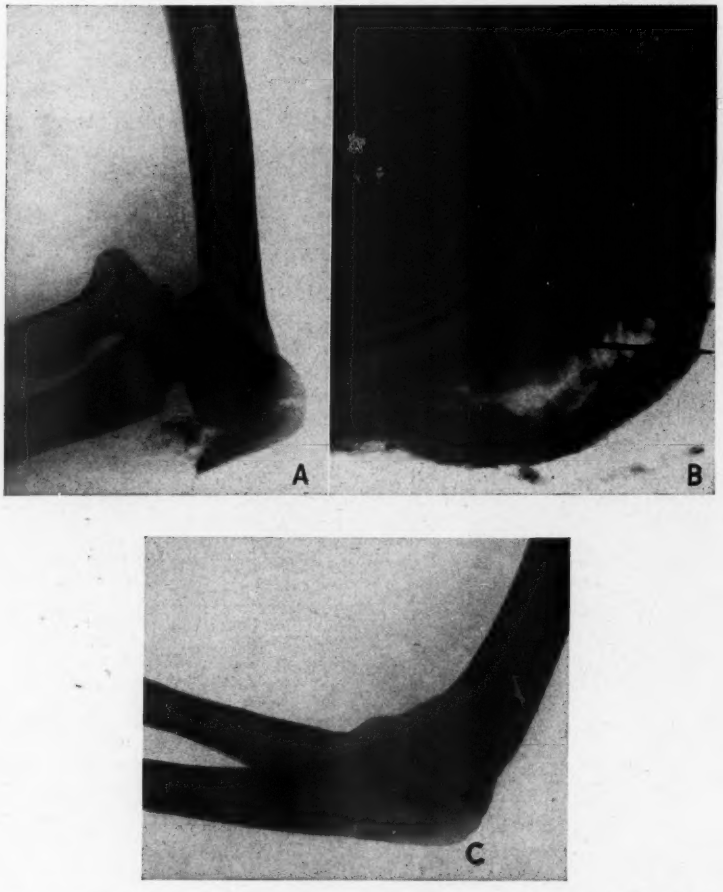

Fig. 4. N. B. W. Male, 47. *A*. Fracture of left olecranon and anterior dislocation of the elbow. *B*. Fracture of olecranon reduced and fragments replaced in their normal position. Olecranon anchored with two crossed Kirschner wires. Wires removed at the end of 4 weeks. *C*. X-ray 2 months after the accident. Olecranon well united in perfect position. Good joint function.

the wires are removed without disturbing the rest of the cast. Only slight force is needed to withdraw the wires and the two tiny holes in the skin heal well without further treatment. A few weeks later, after the fracture is well united, the cast is removed and exercises of the joints begun.

I have used the crossed Kirschner wires in twelve cases which have involved fractures of the condyles of the tibia and femur, patella, malleoli of the ankle and olecranon. All but one have healed well with good function. The only failure was in an early case of T-fracture of the lower end of the femur in a very heavy man. Due to technical errors in the operation, the wires were not properly placed to hold the fragments. In every one of these cases I have been impressed with the security of attachment which the crossed wires afford. Thus in fractures of the patella and olecranon where

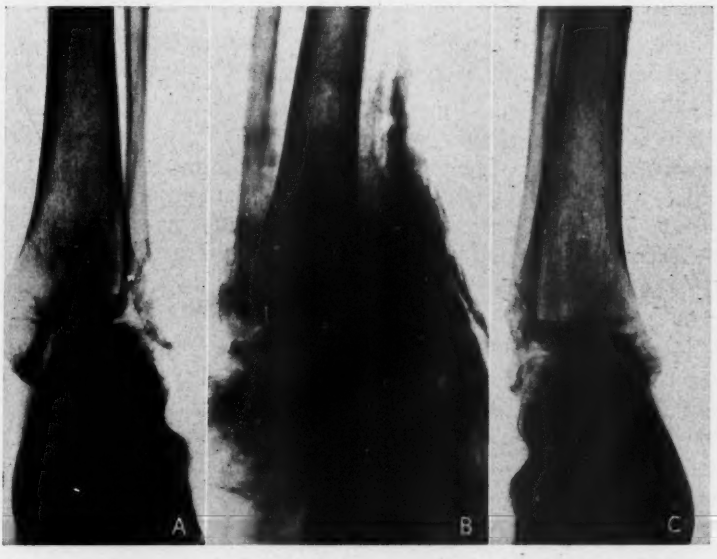

Fig. 5. W. P. Male, 25. *A*. Old malunited fracture of the left ankle with foot displaced laterally. *B*. X-ray after fracture was reduced. Crossed Kischner wires drilled through internal malleolus into the shaft of the tibia, thus locking the fragments securely. Wires removed at the end of 4 weeks. Cast removed at 6 weeks. *C*. X-ray two months after the operation showing perfect position of the astragalus beneath the tibia. Malleoli solidly united in their normal position. No residual disability.

strong forces tend to separate the fragments, the wires have anchored them firmly together. Moreover, in old fractures wherein the bones were osteoporotic and soft, these wires have secured a strong enough hold to resist any tendency to displacement.

In conclusion, this method of fixation of fractures is recommended because of its utter simplicity, its great efficiency, and because no metal appliances are left in the bone which require subsequent surgical removal.

REFERENCE

1. Venable, C. S.; Stuck, W. G., and Beach, Asa: The Effects on Bone of the Presence of Metals Based Upon Electrolysis, Ann. Surg. *105:* 917 (June) 1937.

PREOPERATIVE USE OF PROTAMINE ZINC INSULINATE IN A CASE OF EXOPHTHALMIC GOITER WITH DIABETES MELLITUS

CLARENCE E. BIRD, M. D., F. A. C. S.
Professor of Surgery, University of Louisville
Louisville, Kentucky

THE purpose of this article is to report a case in which preparation for operation was made simpler and more advantageous to the patient by the substitution of protamine zinc insulin for regular insulin.

The problem presented was to build up weight and strength in an emaciated, iodine-fast patient with exophthalmic goiter and moderately severe diabetes mellitus. This was accomplished, in brief, by rest in bed, the withdrawal of stimulants, the withdrawal of iodine for six weeks, and the provision of a high caloric diet covered by insulin. The final preparation by iodine and the subsequent operative procedures were then carried out with ease.

The large doses of regular insulin which were given during the first four weeks following admission caused anxiety to both patient and doctors because of frequent rather severe hypoglycemic reactions. A change to protamine insulin reduced the number of injections, allayed the fears of the patient, stopped the reactions, and improved the control of the glycosuria and acetonuria.

The accompanying table gives all of the detailed information which is of interest in the case.

REPORT OF CASE

An American housewife, aged 29, was admitted to the Louisville City Hospital on March 3, 1937, with the complaints of goiter, exophthalmos, nervousness, excessive perspiration, pounding of the heart, dyspnea, and loss of weight in spite of a ravenous appetite. The goiter had enlarged gradually over a period of seven years, but the major symptoms had become noticeable only since October, 1936. In January, 1937, her physician prescribed Lugol's solution, 5 drops 3 times a day, later increased to 10 drops 3 times a day, on which she gained four pounds in weight, from a January low of 74 pounds to an admission weight of 78 pounds. Her previous average weight had been 113 pounds. There had been an occasional sensation of pressure on the trachea, but no change in the voice. For several months she had drunk large amounts of water and passed urine copiously. The menstrual periods had been normal until September, 1936, when absolute amenorrhea began. Headaches and failing vision had not been noted.

From the Department of Surgery, University of Louisville.

Examination showed exophthalmos, emaciation, excitation, hyperhydrosis, choreiform movements and fine tremor of the hands. Fundi oculi were normal. There was dental caries. The thyroid gland was bilaterally enlarged, diffusely lobulated and elastic, with prominent veins and pulsating small arteries over the anterior surface of the organ. The thrill over the right upper pole was forceful, and loud bruits were heard over the whole gland. The ribs were prominent, the breasts atrophic, and the thorax was shaken by a hyperactive heart; rate 132, rhythm regular with a loud, harsh systolic murmur, loudest over the mitral area but heard over the whole left chest and in the left axilla. The systolic arterial pressure was 140, the diastolic 80. The lungs were normal. The liver was not palpable and there was no edema.

Laboratory determinations: Red blood count, 5 million; hemoglobin, 85 per cent; white blood count, 6,600; urine, specific gravity 1.040, sugar 4 plus, acetone 1 plus, sediment normal; blood Kahn negative. A roentgenogram of the chest showed normal lungs, a small heart of normal contour, and no evidence of substernal thyroid. Films of the skull showed a normal but rather small pituitary fossa. An electrocardiogram indicated sinus tachycardia, rate 140.

The diagnoses were:

Goiter, diffuse, with hyperthyroidism (thyrotoxicosis);

Diabetes mellitus.

The detailed preoperative care of the patient is outlined in the table, and discussed later on. On April 29, 1937, eight weeks after admission to the hospital, a subtotal resection of the right lobe and isthmus of the thyroid gland was carried out under morphine-atropine-nembutal-cyclopropane-oxygen anesthesia. Silk was used and the wound was closed without drainage. The homogeneous mass of tan-red, firm, diffusely nodular tissue measured 8 by 4 by 3.5 cm. Microscopic examination showed diffuse hyperplasia modified by iodinization. Subtotal resection of the left lobe was accomplished without incident thirteen days later. The same technic was used, except that drainage was necessary. The patient was discharged from the hospital, improved, May 26, 1937, twelve weeks after admission.

Discussion

In October, 1935, Scott and Fisher[1] reported that in rabbits injected with insulin containing 0.1 per cent of zinc, the physiologic action of the insulin was much delayed and the level of the blood sugar remained considerably below normal ten hours after commencing the test. The quantity of sugar metabolized due to the zinc-insulin solutions was at least equivalent to that observed with standard solutions of insulin. The effect was delayed and prolonged, but the total activity was undiminished. The work was confirmed for normal dogs by Kerr, Best, Campbell and Fletcher[2] and by Fazekas and Himwich.[3] Scott and Fisher[4,5] later suggested, based again on experiments on rabbits, "that zinc, or some other metal, is largely responsible for the combination that results in the prolonged hypoglycemia produced by insulin with protamine."

Date	Diet in Calories	Added Calories as Glucose	URINE						INSULIN		
			7AM-NOON		NOON-5PM		5PM-7AM		A. M.	NOON	P. M
			Sugar	Acetone	Sugar	Acetone	Sugar	Acetone			
3/3	Regular	.....	0	0	0	0	0	0	0	0	0
4	3500	.....	4	1	0	0	0	0	0	0	0
5	....	80 p.o.	0	0	0	0	4	1	10r	20r	20r
6	....	320 p.o.	0	0	4	1	0	0	20r	20r	40r
7	....	.....	0	0	4	0	4	0	10r	20r	20r
8	....	.....	4	1	4	1	4	1	25r	30r	25r
9	....	.....	4	1	3	1	3	1	25r	25r	25r
10	....	.....	2	1	0	0	1	0	30r	30r	30r
11	....	.....	0	0	3	1	2	1	30r	30r	30r
12	....	.....	3	0	3	1	3	0	30r	30r	30r
13	....	.....	4	1	2	0	0	0	35r	40r	40r
14	....	.....	3	1	0	0	2	0	35r	40r	40r
15	....	.....	3	1	4	1	3	1	35r	40r	40r
16	....	.....	1	0	4	2	0	0	35r	40r	40r
17	....	.....	0	0	0	0	0	0	40r	45r	45r
18	....	.....	0	0	0	0	0	0	40r	45r	45r
19	....	.....	2	1	4	2	3	1	40r	45r	45r
20	....	40 i.v.	2	0	0	0	0	0	40r	45r	45r
21	....	80	0	0	3	0	0	0	40r	45r	45r
22	....	120 p.o.	1	1	4	1	0	0	40r	45r	40r
23	4000	140 i.v.	0	0	2	0	0	0	40r	45r	40r
24	....	110	2	0	3	1	1	0	40r	45r	0
25	....	280 p.o.	4	0	4	0	3	0	30r	35r	30r
26	....	280 p.o.	4	0	4	0	0	0	30r	35r	30r
27	....	280 p.o.	4	0	2	0	0	0	35r	40r	35r
28	....	280 p.o.	0	0	0	0	0	0	35r	40r	35r
29	....	280 p.o.	3	0	4	0	1	0	35r	40r	35r
30	....	280 p.o.	2	0	4	0	0	0	35r	40r	35r
31	....	80 p.o.	4	0	3	0	0	0	35r	40r	35r
4/1	....	80 p.o.	4	0	3	0	0	0	55	20p	15p
2	....	.....	0	0	2	0	0	0	20p	20p	15p
3	....	.....	0	0	3	0	2	0	45p	0	15p
4	....	.....	4	0	3	0	1	0	50p	0	20p
5	....	.....	0	0	2	0	4	0	60p	0	20p
6	....	80 p.o.	0	0	3	0	4	0	70p	0	20p
7	....	40 p.o.	0	0	4	0	0	0	80p	0	20p
8	....	40 p.o.	0	0	3	0	3	0	90p	0	20p
9	....	.....	0	0	0	0	0	0	90p	0	20p
10	....	.....	0	0	1	0	1	0	100p	0	0
11	....	.....	0	0	1	0	0	0	100p	0	0
12	....	.....	1	0	2	0	1	0	100p	0	0
13	....	.....	1	0	0	0	0	0	100p	0	0

p.o., by mouth; i.v., intravenously.
r—indicates regular insulin.
p—indicates protamine zinc insulinate.

Date	Blood Sugar	B. M. R.	Pulse	Weight	REMARKS
3/3	...	.....	140	..	
4	...	.....	130	..	
5	...	+ 56	120	78	
6	...	.....	120	..	
7	...	.....	124	..	
8	200	.....	130	..	
9	...	+ 83	124	81	
10	...	.....	118	..	
11	...	.....	120	..	
12	...	.....	118	..	
13	...	.....	112	..	
14	...	.....	106	..	
15	...	.....	110	..	
16	...	+ 81	118	82	
17	181	.....	112	..	
18	...	.....	114	..	
19	...	+110	110	82	
20	...	.....	108	..	*H. R. in p. m.
21	...	.....	120	..	*H. R. in p. m. Half of glucose by mouth.
22	...	.....	116	..	*H. R. in p. m.
23	...	+ 75	114	83	*H. R. in p. m. Nauseated.
24	...	.....	116	..	*H. R. in p. m. Two-thirds glucose p. o.
25	...	.....	110	..	
26	...	+ 81	106	83	
27	285	.....	92	..	
28	...	.....	90	..	
29	...	.....	106	..	
30	...	+100	102	84	
31	...	.....	90	..	
4/1	...	.....	92	..	The a. m. insulin was 35r, 20p.
2	70	+ 75	102	87	
3	40	.....	104	..	Insulin 7 a. m. and 4 p. m. *H. R. in a.m.
4	...	.....	100	..	
5	...	.....	96	..	Second insulin, 11 p. m.
6	...	.....	100	..	
7	...	.....	106	..	Slight *H. R. in a. m.
8	...	.....	104	..	
9	50	.....	104	..	
10	...	.....	104	..	
11	...	.....	100	..	Insulin at 5 a. m.
12	67**	.....	102	..	
13	...	+ 69	100	89	Lugol's solution instituted, 10 drops t.i.d.

*H. R. stands for Hypoglycemic Reaction.
**Subsequent blood sugars on the same day were 181 and 222.

Date	Diet in Calories	Added Calories as Glucose	URINE 7AM-NOON Sugar	7AM-NOON Acetone	NOON-5PM Sugar	NOON-5PM Acetone	5PM-7AM Sugar	5PM-7AM Acetone	INSULIN A. M.	INSULIN NOON	INSULIN P. M
14	....	80 p.o.	3	0	1	0	2	0	100p	0	0
15	4000	.....	1	0	3	0	4	2	100p	...	...
16	....	.....	0	0	0	0	0	0	100p	...	...
17	....	.....	0	0	2	0	..	..	100p	...	...
18	....	.....	0	0	1	0	3	0	100p	...	...
19	....	.....	1	0	3	0	4	0	100p	...	...
20	....	.....	3	0	2	..	3	..	100p	...	...
21	....	.....	2	0	3	0	4	0	110p	...	...
22	....	.....	3	0	3	0	4	0	110p	...	...
23	....	.....	0	0	0	0	1	0	110p	...	...
24	....	.....	2	0	1	0	1	..	110p	...	...
25	....	.....	2	..	4	..	3	..	110p	...	...
26	....	.....	4	..	3	..	4	..	110p	...	...
27	....	.....	0	..	2	..	2	..	110p	...	...
28	....	20 p.o.	2	..	2	..	1	..	100p	...	...
29	220	200 i.v.	3	2	2	1	0	0	0	25r	20r
30	2600	160 p.o.	0	0	0	0	0	0	10r	10r	10r
5/1	3500	80 p.o.	0	0	0	0	0	0	20p	...	...
2	....	160 p.o.	1	0	4	2	2	1	20p	10r	10r
3	....	160 p.o.	2	0	3	1	3	1	30p	...	...
4	....	160 p.o.	1	0	1	0	2	1	30p	...	...
5	....	160 p.o.	1	1	3	0	4	0	30p	...	...
6	....	160 p.o.	4	0	3	0	0	0	30p	...	...
7	....	80 p.o.	3	1	4	1	4	1	45p	...	...
8	....	80 p.o.	1	0	4	0	4	0	45p	...	...
9	....	80 p.o.	0	0	2	0	4	0	45p	...	...
10	....	80 p.o.	2	0	2	0	4	0	45p	...	...
11	....	80 p.o.	0	0	1	0	4	2	45p	...	...
12	80	200 i.v.	0	0	4	1	3	0	25p	25r	20r
13	1156	40 p.o.	0	0	0	0	0	0	25r	25r	25r
14	....	.....	0	0	0	0	0	0	25r	25r	25r
15	1314	.....	2	0	2	0	0	0	25r	25r	25r
16	....	.....	0	0	3	1	2	0	25r	25r	25r
17	1558	.....	2	0	2	1	0	0	25r	25r	25r
18	....	.....	0	0	0	0	2	0	25r	25r	25r
19	1669	.....	0	0	1	0	2	0	25r	25r	25r
20	....	.....	0	0	2	0	3	0	25r	25r	25r
21	....	.....	0	0	2	0	2	0	25r	25r	25r
22	....	.....	0	0	0	0	0	0	25r	25r	25r
23	....	.....	1	0	1	0	3	0	25r	25r	25r
24	....	.....	0	0	3	0	2	0	25r	25r	25r
25	1808	.....	0	0	2	0	..	..	30r	30r	30r
26	....	.....	0	0	0	0	0	0	30r	30r	30r

DATE	BLOOD SUGAR	B. M. R.	PULSE	WEIGHT	REMARKS
14	40	.....	110	..	
15	...	.....	104	..	
16	...	+ 95	100	89	
17	82	.....	100	..	Lugol's solution, 15 drops t.i.d.
18	...	.....	100	..	
19	...	.....	100	..	
20	...	+ 46	100	89	
21	125	.....	92	..	
22	93*	.....	96	..	
23	...	+ 62	100	90	
24	...	.....	98	..	
25	...	.....	108	..	
26	...	.....	108	..	
27	...	+ 48	100	94	
28	...	.....	102	..	
29	100**	.....	120	..	RIGHT LOBECTOMY. ***
30	...	.....	116	..	Lugol's solution drops 15 t.i.d.
5/1	...	.....	126	..	
2	...	.....	118	..	
3	298	.....	118	..	
4	...	.....	120	..	
5	...	.....	110	..	
6	...	.....	110	..	
7	...	.....	120	..	
8	...	.....	120	..	
9	...	.....	102	..	
10	222	.....	102	..	
11	...	+ 13	100	93	
12	...	.....	120	..	LEFT LOBECTOMY. ***
13	80	.....	110	..	Lugol's solution, 15 drops t.i.d.
14	...	.....	120	..	
15	...	.....	100	..	
16	...	.....	94	..	
17	160	.....	94	..	
18	...	.....	90	..	
19	...	.....	82	..	
20	...	.....	82	..	
21	...	+ 20	84	94	
22	...	.....	84	..	
23	...	.....	88	..	
24	153	.....	90	..	
25	...	.....	88	..	
26	...	.....	94	..	

*Subsequent blood sugars on the same day were 166, 250 and 181.
**Postoperative blood sugar 181.
***Lugol's, 15 drops 4 times per rectum.

Combining the work of the Danish group,[6] who introduced protamine insulin, with these observations of Scott and Fisher, manufacturers have made available a protamine-zinc-insulinate which, compared to the original protamine insulin, has a higher concentration of zinc, and hence a slower and more prolonged effect. That the protracted activity of these modern insulins is due to slow absorption from the tissues in which they are deposited was shown indirectly by Longwell and Ravin[7] when they noted in rabbits that if introduced intravenously, regular and protamine insulins act with equal rapidity. The slower absorption of protamine insulin from the tissues, originally hypothesized by the Danish investigators,[6] has been beautifully confirmed by direct observations on rabbits by Beecher and Krogh.[8]

The chief advantages of protamine insulin over regular insulin were first worked out from a clinical standpoint by Hagedorn, Jensen, Krarup and Wodstrup,[6] Root, White, Marble and Stotz,[9] Kerr, Best, Campbell and Fletcher,[2] Sprague, Blum, Osterberg, Kepler and Wilder,[10] and Lawrence and Archer.[11] These advantages are: (1) that the hyperglycemia, urinary sugar and acidosis are more uniformly controlled, considering the 24-hour period as a whole; (2) that there are fewer hypoglycemic reactions, and those that occur are milder and of more gradual onset than with regular insulin; (3) that the number of injections per day is decreased; and (4) that, due partly to the last factor and partly to metabolic efficiency, the sense of well-being and the capacity for work of the patient are improved.

The first of these advantages is the most important and the one on which all the others depend. It is an especially important factor in severe diabetes, juvenile diabetes, and in cases of excessive metabolic activity. For this reason the new insulin was thought to be particularly appropriate for use in a case of diffuse goiter with hyperthyroidism such as the one here reported.

This patient was iodine-fast on admission. Iodine was withdrawn therefore for six weeks, and the time was made use of to improve the general condition and psychologic outlook of the patient. A high caloric intake was necessary to offset the excessive metabolism and provide additional calories for gain in weight. Examination of the table shows that on a 3,500 calorie diet, 130 units of regular insulin in divided doses were necessary to control glycosuria and acetonuria, and that the control was even then imperfect. From March 20 to 24, there were severe and unpredictable hypoglycemic reactions. These were prevented after three days by increasing the calories to 4,000 and decreasing the total regular insulin dosage

to 95 units. However, the glycosuria became more pronounced between March 25 and April 1.

A transition to treatment with protamine zinc insulin was then made, at the same time maintaining the 4,000 calorie diet. Satisfactory control of the glycosuria and almost complete avoidance of reactions resulted. It was found that a single dose of 110 units of protamine zinc insulin given subcutaneously an hour before breakfast worked out well. On April 22, a day on which the urinary sugar was not quite as well controlled as on most other days, four blood sugar determinations taken during the 24 hours showed 93, 166, 250 and 181 milligrams per 100 cubic centimeters. On April 9, and on April 14, when blood sugar determinations showed values of 50 and 40 mg., respectively, there were no reactions. When the patient became confident that she would have no more reactions, she became much more calm in spirit.

As may be seen on the chart, the weight increased progressively from the time of admission to the time of the first operation, with a gain of 16 pounds in eight weeks. During the first six weeks, while the patient was given no iodine, the basal metabolic rate varied between plus 56 and plus 110. The pulse rate decreased gradually from 130 to 100. Lugol's solution given 10 to 15 drops three times a day for two weeks made very little change in the basal metabolic rate or in the pulse rate, but both were within satisfactory limits and the patient appeared particularly well at the time of the first operation.

The insulin requirement dropped from 100 units of protamine zinc insulin for 4,000 calories, to 45 units for 3,500 calories, following the removal of the right lobe and isthmus. After the excision of the left lobe the caloric intake was decreased and regular insulin was used, of which 75 units a day seemed to be required on an 1,800 calorie diet. The basal metabolic rate was plus 13 after the first operation, and plus 20, nine days after the second. The pulse rate receded to between 82 and 94 after the final operation.

Conclusion

Protamine zinc insulinate was found to be advantageous in the management of this patient whose diabetes was aggravated by diffuse goiter with hyperthyroidism.

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ACUTE PHLEGMONOUS AND NON-TRAUMATIC PERFORATIVE LESIONS OF THE COLON

Report of Three Cases with Intraperineal Hemorrhage Complicating One

GEORGE H. BUNCH, M. D.
Columbia, S. C.

BECAUSE of its physical condition and function the colon is for most of us an uninviting field for study. This explains much of our ignorance of it and of its diseases. It also explains our failure to examine it in the individual patient unless clinical symptoms definitely point to it as the seat of disease. Its physiology is not well understood. That it is more than a terminal conduit for intestinal refuse is proven by the many glands in its mucous membrane. Of all the viscera the colon is unique in that, although the small intestine reaches its full length at the age of 12, the colon "continues throughout life to increase slowly in length actually and in relation to the length of the small intestine."[1] As its lumen gets smaller from the cecum to the ampulla of the rectum the bacterial content of the material passing through it greatly increases. Maintaining proper hygiene and flora of the colon is of practical importance. Most infectious colitis may be prevented by giving lactic acid organisms routinely in the food. Buttermilk every day keeps the doctor away. We do not understand the normal resistance of the colon to the many pathogenic bacteria that it harbors. This is relatively small in infancy and increases as immunity is acquired with age. The quality is innate and when impaired inflammation and ulceration occur.

The deadliness of infectious colitis even in adults was shown in our Civil War in which more soldiers died of it than of wounds. Andrew Jackson could hardly ride his horse at the battle of New Orleans because of weakness from it. Marion Sims moved to New York from Montgomery because of ill health from it and he describes in his autobiography how Doctor McClellan, Professor of Surgery at Jefferson Medical College, died of acute perforation of the colon in 1847, probably the first case recorded in American literature.[2]

The diagnosis and the treatment of colitis and of colonic ulcer are problems for the proctologist and the clinician. Acute phlegmonous inflammation and acute perforation of the colon, however, are tragedies which, though rare, are real surgical emergencies. In an active work of 20 years my experience has been limited to four such cases. The diagnosis in none had been suspected before laparotomy.

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At operation each presented problems in judgment and in management which together have been made the basis of this paper.

Report of Cases

Case 1: A white man, aged 36, was admitted complaining of abdominal pain and nausea. The temperature was 101; respirations 22, pulse 98. The leukocytes were 8,000 with 89 per cent polymorphonuclears. The abdomen was distended and generally tender, without rigidity. There was a wide weak scar from a McBurney incision made in boyhood. He had been in good health until 36 hours before admission. At that time he had been seized with pain about the navel. Being constipated he took a laxative. An enema was discontinued because of pain.

At operation done through the appendectomy scar for the relief of incisional hernia with incomplete intestinal obstruction, omental hernia with universal fibrous adhesions of the small gut in this region was found. In addition there was diffuse peritonitis with serosanguinous exudate and plastic lymph bathing the viscera and the non adherent intestine. This was without colonic odor. Through a right rectus incision the duodenum, stomach, gallbladder and pancreas were found to be normal except for peritonitis. Palpation and visualization, where possible, of the intestine failed to show any lesion that might have caused peritonitis. The pelvis was drained and the abdomen closed, the diagnosis being peritonitis of unknown origin. A culture of the exudate was not made. The patient ran a septic postoperative fever for 16 days before death.

At autopsy there were found several acute punched-out ulcers of the lower sigmoid with perforation of a large ulcer at the peritoneal reflexion near the rectosigmoid junction.

Comment: This patient died of peritonitis from perforation, of a simple ulcer of the upper rectum, which must have been overlooked at operation. Non-malignant ulcers of the colon may complicate systemic disease, may follow thrombosis or embolism of the small arteries of the gut wall or may be of trophic origin. In tuberculosis, syphilis, amebic dysentery and typhoid fever they are caused by the specific organism. Many ulcers result from infection, often of a mixed type, by unidentified organisms. Chronic ulcerative colitis is a group of such unclassified lesions which will ultimately be subdivided.

Barron has already noted that ulcers with marked tendencies to perforate have distinctive characteristics. In 1928 he reported three cases of simple non-specific ulcer of the colon with 50 cases from the literature[3]. Later he had a fourth case[4]. In 1931 Wise reported 2 cases[5], and in 1934 another 4 cases with 2 additional cases from the literature[6], making a total of 62 cases. Of the 58 cases included in Wise's 1931 study the portions of the large gut involved with the mortality of the perforated and the non-perforated cases in each is shown in Table 1.

TABLE 1

	Perforated Cases	Died	Re-covered	Non Perforated Cases	Died	Re-covered
Cecum	11	7	4	4	1	3
Ascending Colon	13	6	7	1	1	0
Hepatic Flexure	4	4	0	0	0	0
Transverse Colon	4	4	0	1	1	0
Descending Colon	1	1	0	1	1	0
Sigmoid	13	9	4	1	1	0
Rectum	2	2	0	2	0	2
Total	48	33	15	10	5	5

In the beginning the lesion, though often multiple, is always acute with more than half the recognized cases perforating. The ulcer is clean cut and penetrating without undermining. It may heal with scar or it may become chronic. The average age of the patient is 40 years. Two thirds of the cases occur in males. Most cases go unsuspected and unrecognized because they are practically symptomless. Constipation is the most common complaint. Except when the ulcer is in the lower rectum or about to perforate pain is not severe and there is but little tendency to bleed. Of the 42 cases in which perforation occurred, in 30 general peritonitis developed and in 12 localized abscess. In none was the diagnosis made before operation except one in which the ulcer was in the rectum. If on the right side the condition is confused with appendicitis. If on the left with malignancy. When low enough to be reached by the sigmoidoscope the ulcer may be visualized, otherwise we are dependent upon the x-ray for diagnosis.

CASE 2: A white man of 35 was admitted complaining of pain in the abdomen and nausea. His temperature was 101; respirations 22, pulse 110, leukocytes 8,000 with 85 per cent polymorphonuclears. The hemoglobin was 65 per cent, the systolic pressure 115, the diastolic 60. He had been in bed on light diet since the onset of pain 48 hours before admission. Except for constipation and vague distress in the left abdomen for several months he had been in good health. The abdomen was distended, with rigidity and muscle spasm more marked on the left. Pallor of the conjunctiva was somewhat greater than was indicated by the blood examination.

Glucose and salt solution were given and exploratory laparotomy under spinal anesthesia through a midline incision was done for spreading peritonitis, probably from a ruptured viscus. Massive hemorrhage was found in the free peritoneal cavity complicating general peritonitis. There had been no history of trauma. The spleen and the liver were normal in size and consistency. There was no fat necrosis and the pancreas was normal. No lesion was found in the stomach, the duodenum or the kidneys. The small intestine was congested and distended from peritonitis but without perforation. The colon was not acutely distended. Near the splenic flexure and covered by the great

omentum the surface of the descending colon for several inches was dark and discolored about a perforated ulcer in the wall. A vein in the necrotic omentum near its attachment to the colon and near the ulcer was bleeding. There was no localization. There was definite colon odor to the extravasated blood in this region. The bleeding vein was ligated and the perforation imperfectly closed, the gut wall being too friable for sutures to hold well. After the blood had been removed from the peritoneal cavity the descending colon was walled off by rubber dam, which was brought out through a stab wound under the left costal margin. A cigarette drain was placed in the pelvis and the incision closed. A transfusion of 500 c.c. of unmodified blood was given. He died in 26 hours.

The autopsy findings were: "The middle portion of the descending colon is the site of an unusual kind of inflammation which involves 5 inches of gut. There is extensive destruction of the mucosa with wide margins of gangrene about suppurative areas in the gut wall. The centers of these gangrenous areas extend through the entire thickness of the wall and have resulted in two large perforations each about an inch in diameter, and in one small perforation of about ¼ inch. The cause of death is acute inflammation of the descending colon with suppuration, ulceration and gangrene of the gut wall, with multiple perforations, intraperitoneal hemorrhage and general peritonitis."

Comment: Spontaneous intraperitoneal hemorrhage complicating perforation of an acute ulcer of the colon makes this a unique case. The primary lesion consisted of multiple simple ulcers of the colon. As these penetrated the gut wall they were portals of entry for organisms, of stercoraceous origin, which caused acute inflammation followed by suppuration, and perforation of the wall with necrosis of the omentum and massive hemorrhage.

Intraperitoneal hemorrhage from tubal pregnancy or ruptured cyst of the ovary is not uncommon in women. In men, however, intraperitoneal hemorrhage not due to trauma or malignancy is rare, only a few cases having been recorded. This is remarkable for bleeding into the stomach and into the intestine is common in ulcer and in other lesions. Arteriosclerosis, cirrhosis of the liver, spontaneous rupture of the spleen or liver and acute pancreatitis are recognized causes of intraperitoneal hemorrhage.

In 1911 Churchman[7] reported a case of fatal spontaneous hemorrhage in a man of 48. In it and in two similar cases from literature he considered hemorrhagic disease the cause. There is experimental evidence of this in animals but none in the human, intraperitoneal hemorrhage having been found in dogs after chloroform poisoning.

In 1925 Bok and Bamforth[8] in a man of 28 with undulant fever, enlarged liver and thrombosis of the portal vein, found the abdomen distended with blood.

Pyrah, Stansfield and Garland[9] have recorded a case of fatal hemoperitoneum due to spontaneous rupture of the splenic vein in

a woman of 38, and Bruce[25] has reported a case of intraperitoneal hemorrhage in a woman of 40 from spontaneous rupture of the liver.

Greene and Powers[27] reported intra-abdominal apoplexy in a woman of 54 who recovered after the bleeding gastroduodenal artery was ligated.

Hartly and MacKechnie[10], in fatal spontaneous intraperitoneal hemorrhage in a man of 31, finding no definite source at autopsy, considered the condition due to "splanchnostaxis."

Buchbinder and Greene[11], in a fourth case of true intraperitoneal apoplexy, state in all four cases the ruptured vessel was a branch of the celiac axis which was ligated at operation with recovery. Three had as a basis hypertension and all four had arteriosclerosis.

In 1935 Thompson and Dumpley[26] discussed abdominal apoplexy finding only eight cases in the literature including the one of their own.

In 1936 Moorehead and McLester[12] had two fatal cases of abdominal apoplexy, one in a man of 44 with ruptured gastric artery and the other in a man of 50 with ruptured superior mesenteric artery. I think abdominal apoplexy, with exploratory laparotomy being so frequently done, will in the future be found more often.

CASE 3: A fat white man of 62 was admitted with temperature 101; pulse 90, respirations 24. The leukocytes were 11,300 with 71 per cent polymorphonuclears. The urine contained pus and casts. He complained of pain in the abdomen, nausea and vomiting beginning 3 days before. He had usually been constipated. There was distention and tenderness of the abdomen definitely greater on the right. No masses could be felt. We thought he had diffuse peritonitis from a perforated appendix. Glucose and salt solution were given. Respirations were kept under 15 per minute by repeated doses of morphine. The stomach was kept empty by an indwelling nasal tube. Next day his temperature reach 104. On the third day after admission there was suggestion of an indefinite mass filling the right side.

At exploratory laparotomy done under spinal anesthesia through a right transverse incision 1 inch above the navel there was found diffuse peritonitis. The great omentum was lightly adherent to the ascending colon which was so distended that it filled the entire right abdomen. The colon was purplish-red except along the anterior portion, the part on the opposite side from the mesenteric attachment. This was gray and mottled, a massive phlegmon of irregular outline, 2 or more inches wide, extending upward to the hepatic flexure and downward toward the cecum as far as could be seen. It was as if there were a series of large confluent carbuncles in the gut wall each with multiple craters. The fact that this was limited to a longitudinal strip of the gut farthest away from the mesentery suggested that it followed thrombosis

of the small terminal vessels, caused by the pressure of acute distention of the colon. Under gentle manipulation the gut wall suddenly gave way and about a quart of liquid feces escaped. The colon collapsed. Because of the poor condition of the patient and because of gross soiling further exploration was not done. The ascending colon was isolated by rubber dam which was brought through the lower angle of the wound, the upper part of which was loosely approximated with interrupted silk worm sutures. The family was told that the patient would soon die.

However, because of the isolation of the colon by the rubber and because of adequate dependent drainage that could have been attained only through a transverse incision, the man improved after operation. The decompression of the colon relieved the distention and the toxemia. With improving appetite and strength he became cheerful. After soft diet was begun on the fifth day his bowels moved daily in the normal way, although there was fecal discharge from the wound. On the twelfth postoperative day there was pain in the right shoulder. On the fifteenth day x-ray examination showed the right diaphragm high, with increased density of the lowest lobe of the right lung, suggesting subphrenic abscess. On the next day, after pus had been obtained by needle, two drainage tubes were inserted into the abscess through an incision made under local anesthesia in the mid axiliary line between the eighth and ninth ribs. The next day he died. Permission for autopsy was refused.

Comment: This was a case of acute diffuse phlegmonous colitis first seen on the third day after onset, with diffuse spreading peritonitis, thought at the time to have come from a perforated appendix. Operation was deferred until the sixth day when it was thought that early localization of the infection had taken place. Had operation been done earlier resection of the diseased colon might have cured the patient. At the time of operation the pathologic process was so extensive that he could not have survived resection. He died on the sixteenth postoperative day of right subphrenic abscess.

Phlegmonous colitis is a rare lesion. Most references to it are in Scandinavian literature which suggests, as the etiologic factor, trauma to the gut lining from fish bones that have been swallowed with the food. In 1915 Von Saar and Gunter[13] could find but 15 cases of fulminating inflammation of the ascending colon.

Hellstrom[14], in a study of primary phlegmon of the intestine, collected 55 cases from literature and added 4 of his own. There has never been a case in which the diagnosis was made before laparotomy or autopsy. The disease has no pathognomonic symptoms and presents no distinctly marked clinical picture within the abdomen. Of the 59 cases studied there were only 2 resections, 1 of these by Dowd, was the only case of the series to recover. Hellstrom concluded that resection for acute phlegmon of the intestine is indicated if it be technically possible.

Bohamson[15], in a paper on phlegmonous enteritis, says all authorities agree as to its rarity and as to the gravity of the prognosis. He distinguishes three types:

1. Diffuse phlegmon of the intestinal wall, most often occurring in the duodenum and upper gut, and caused by streptococci.

2. Solitary or multiple phlegmon.

3. Chronic inflammatory phlegmon or pseudo-tumor which usually occurs in the colon. The type of inflammation often depends upon the flora present in the involved gut for the infection is usually of enteric origin although it may come by the blood stream. A mechanical injury may be an entrance for infection. In the acute progressive cases the streptococcus is the prevailing organism. In his compilation the portion of gut is shown in Table 2.

TABLE 2

	No. Cases	*Per Cent*
Entire gastrointestinal tract	1	1.5
Duodenum	18	26.5
One part of small intestine	19	28.0
Colon	30	44.0

Irwin and McDonald[16] have made the only comprehensive study of phlegmonous infection of the intestine found in the English language.

I have assembled only 3 cases of recovery after resection of the colon; 1 by Dowd[28] of the descending colon, 1 by Von Saar[13] of the ascending colon by exteriorization and cautery, and the third by Elving[19] of the sigmoid by electrocautery in a man of 56.

CASE 4: A white man of 36 was admitted complaining of pain in the lower abdomen of increasing severity since onset the day before. There had been nausea and vomiting. Both sides were affected equally. There had been no previous attacks. His health had been good. He had been operated upon for an abscess of the rectum two years before. His temperature was 99.5; pulse 125, respirations 25. His leukocytes were 19,200 with 90 per cent polymorphonuclears. On physical examination there was tenderness and some rigidity over the lower abdomen, most marked in the midline above the pubis. No masses could be felt. Immediate operation was done through a McBurney incision for acute appendicitis. The atrophic appendix was not involved. A midline incision was made. The sigmoid was hard and indurated, a sausage-shaped inflammatory tumor 4 inches long, the size of a man's wrist. It was bathed in cloudy exudate containing plastic lymph. There was no free pus. The mass was enclosed in rubber dam placed along the gut on each side and brought through a stab wound. The incisions were closed. He had fever for four weeks before dismissal. His health remained good and he was free

from symptoms, four and a half years after operation. However, on March 3, 1937, he was brought into the hospital with general peritonitis from which he died unoperated upon next day. At autopsy there were multiple diverticula of the sigmoid containing dried fecal masses. He died from an acute perforation of one of these.

Comment: This patient had acute phlegmonous inflammation of the sigmoid from acute diverticulitis which occurs most often in fat men of middle age. From the symptoms it has been called left-sided appendicitis. The inflammation tends to become chronic. Perforation occurs in comparatively few cases. The inflammatory mass has to be distinguished from malignancy. In his case of resection of the sigmoid Dowd[28] described the gut as feeling like a garden hose. Most of the phlegmonous cases in the sigmoid are from diverticulitis. They are the cases best suited for resection, although treated conservatively they tend to resolution and spontaneous relief. In them infection from within the gut, gaining entrance beneath the mucosa, extends along the tissue planes both longitudinally and circularly so that involvement is segmental. This differs from case 3 in which fulminating phlegmonous infection followed circulatory disturbance in the gut wall and extended longitudinally without annular involvement. Such a case tends to necrosis and perforation rather than to chronicity.

Conclusion

In this paper I have presented 2 cases of acute perforation of simple ulcer of the colon. The first was overlooked at exploratory operation for incisional hernia and chronic intestinal obstruction. The second was complicated by phlegmonous colitis and massive spontaneous intraperitoneal hemorrhage. In neither was resection attempted. Both patients died of peritonitis. I have also presented two cases of acute phlegmonous colitis, one a fulminating infection of circulatory origin followed by subphrenic abscess and death, the other an inflammatory tumor of the sigmoid from diverticulitis which recovered after simple drainage of the pelvis but died 4½ years later of peritonitis from acute perforation. All 4 patients were white men previously in good health.

Note: These four men were admitted to Columbia Hospital or South Carolina Baptist Hospital, June 27, 1932, July 16, 1936, Aug. 28, 1936, and Nov. 11, 1932, respectively.

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ANEMIAS OF PREGNANCY

A Review and Report of a Case of the Macrocytic Type With Purpuric Manifestations and Malaria

C. Raymond Mays, M. D.
Shreveport

IT is obvious that the pregnant woman may become anemic because of complications or intercurrent disease. The management of such a case resolves itself into attempting to remove the exciting cause and combating the blood depletion by transfusion or other more or less specific measures. Pregnancy may also be incidental to unusual anemias such as aplastic or sickle cell anemia.

The pregnant woman may also develop anemia, usually moderate but occasionally severe, that more or less depends upon the pregnancy itself as an etiologic factor. For our present purpose the following types may be recognized:

1. Physiologic anemia, uniformly present.
2. Hypochromic microcytic, frequent.
3. Macrocytic (pernicious-like), infrequent.
4. Secondary anemia, from sepsis, hemorrhage, etc.

The last type merits no particular discussion. The other types because of their apparently obscure etiology merit special consideration.

Physiologic Anemia

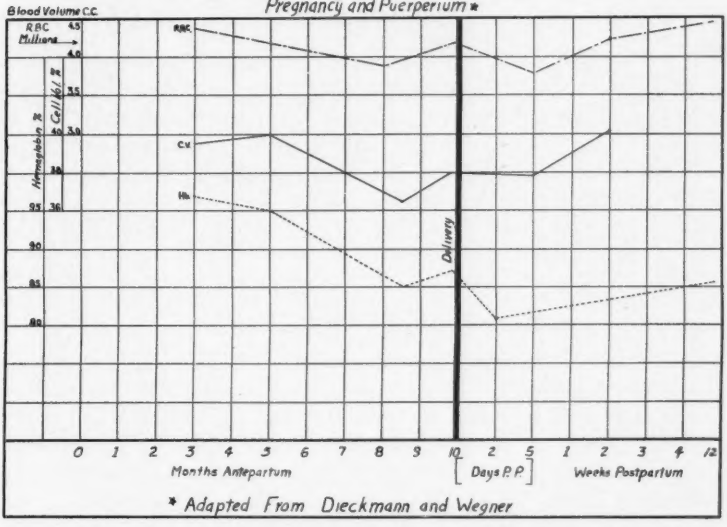
Many observations, notably those of Bland[1], Strauss[2], and Dieckmann[3], have established that the pregnant woman tends to show lower hemoglobin and cell concentration in various periods of pregnancy. A satisfactory portrayal of these changes is illustrated in Chart 1, which shows average values in a group of women observed throughout the pregnancy and puerperium. The puerperal figures are taken from a series other than that which supplied the prenatal values; this gives rise to some degree of inaccuracy but shows the normal trend.

At the twelfth week the hemoglobin is subnormal (14.3 Gm. per 100 c.c. of blood is considered 100 per cent). It decreases to 15 per cent below normal at the twenty-sixth to thirty-fifth antepartum week. It increases in the last week of gestation. Postpartum there is a gradual recovery, but at eight weeks it is still about 14 per cent subnormal.

The erythrocytes follow a similar change in concentration, reaching a decrease of 14 per cent in the third trimester with an immediate prenatal rise. Postpartum, the recovery in erythrocytes is more rapid than the hemoglobin, indicating, as Dieckmann[4] says, "that

the patient is able to produce or maintain erythrocytes in the puerperium, but unable to give them their normal amount of hemoglobin."

Chart 1
Red Cells and Hemaglobin of Blood in Pregnancy and Puerperium *



* Adapted From Dieckmann and Wegner

Further observations made by Dieckmann[4] indicate that though the concentration of hemoglobin and erythrocytes decrease during pregnancy, the total cell and plasma mass increases. The plasma and total blood mass showed an increase of 23 per cent and 25 per cent respectively in the last trimester. Though I have been unable to reconcile entirely the blood volume studies with the concentration curve, the evidence demonstrates that there does occur a total increase in both cell and plasma masses, giving rise to an "oligocythemic hypervolemia." There is relatively a greater increase in plasma than in cells. Certain observations indicate that the increased hydration of the blood in pregnancy follows inversely the protein concentration[5,6]. Dieckmann[7] supposes that the increased flow of blood of lowered viscosity is a mechanism conducive to efficient interchange of gases between mother and fetus.* His blood volume

*At the meeting of the Staff of the Shreveport Charity Hospital, May 18, 1937, after submitting this paper, the author had an opportunity to attend an enlightening symposium on arteriovenous aneurysm. In that the hemodynamic phenomena of this condition are in many respects parallel to those noted in pregnancy a number of interesting questions arise; (1) Can it be assumed that the placenta is, in effect, an arteriovenous fistula which anatomically it is? (2) Is heart enlargement, increased heart output, low blood pressure and the increased plasma volume affected in the pregnant patient because of the placental circulation in the same manner that it is produced in the patient with an arteriovenous aneurysm? (3) Does the pregnant cardiopath do poorly because of increased heart work and lowered coronary pressure? (4) Is the commonly observed puerpal bradycardia analogous to Branham's phenomenon? (5) Are certain sudden unexplained deaths on the delivery bed a result of an acute dilation of a heart already under stress because of the pathologic circulatory changes of even normal pregnancy?

If these questions can be answered in the affirmative it would seem that the increased blood volume (plasma element) in pregnancy is necessary to fill the peripheral vessels because of added placental circulation somewhat as suggested by DeLee.

studies further indicate that after pregnancy the mother retains a great part of her pregnancy gain in total blood quantity.

Dieckmann has also shown that in normal pregnancy the patient has a marked variation in the several indices having (most prominently at the period of greatest dilution) a tendency to large hyperchromic cells. This gives rise to a picture that borders on pernicious anemia[5].

It becomes obvious from these observations that the pregnant woman has lower hemoglobin and cell concentration than her non-pregnant sister. "There is no anemia in the sense of blood loss or failure of blood production"[6]. The mean values for hemoglobin and cell concentration in pregnancy are 11.56 Gm. per 100 c.c. for hemoglobin, 37.31 per cent for cell volume and 3.77 millions per c. mm. for erythrocytes[8].

"The physiologic alteration in blood concentration in pregnancy cannot be altered by treatment of individual cases."[8]

Hypochromic Microcytic Anemia

Hypochromic anemias occur in 30 to 40 per cent of all pregnant women[9]. Because of the disparity of these figures with Adair's[8] (11.6 per cent), it is presumptive that Strauss used non-pregnant values in arriving at the diagnosis of anemia.

Before hazarding a diagnosis of anemia, one should observe the blood count, hemoglobin and cell volume on at least two occasions, because of the wide fluctuation of blood concentration in normal pregnancy. When on repeated examinations, one finds a degree of anemia below the mean average for pregnancy one may entertain the diagnosis of hypochromic anemia. As the name implies, hypochromic anemia is essentially a result of iron deficiency.

Several physiologic phenomena operate to cause iron deficiency in the gravid woman. Strauss[9] postulates that since the growing fetus must withdraw its hemoglobin from the mother, that pregnancy is equivalent to a chronic blood loss. Clough[10] gives pregnancy as an important factor in the production of hypochromic anemia in women. In studies of iron metabolism in pregnancy, Coons[11] has shown that the fetus demands little iron of the mother during the first trimester of pregnancy. The greatest fetal demand is in the last trimester when it requires 4.7 mg. of iron daily and at term requires 8 mg. per day. Because of the relative anoxic state of the fetus, it maintains a plethora and therefore exacts an appreciable demand on the mother for iron. Coons estimates that increase of maternal tissue, demanding blood supply, also adds additional stress on iron metabolism. Because of digestive disturbances, vom-

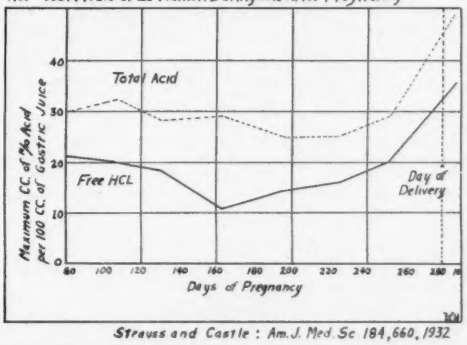
iting, capricious appetite and economic stress, the gravid woman becomes liable to iron deficiency from insufficient ingestion of iron-containing foods such as vegetables and red meats, especially the latter. Richter[12] has observed that if women are given hemapoietic medication supplementary to their diets there is less fall in hemoglobin concentration than is shown in control groups, though the trend remains unchanged.

It has further been observed that women developing hypochromic anemia in the absence of gastrointestinal disturbance invariably give a history of defective diets[13].

The gravida is rendered liable to iron deficiency because of inability to utilize iron, even if sufficient quantity is ingested. Efficient utilization of iron necessitates adequate gastric hydrochloric acid secretion[14], hydrochloric acid being a necessary catalyst for efficient absorption and utilization of iron. Eight per cent of women studied by Strauss[13] during pregnancy had lower concentration of gastric hydrochloric acid than after delivery, and only one third the quantity at six months uterogestation as in the puerperium (Chart II).

Chart 2

Composite Curve of Gastric Secretions Following Alcohol Test Meals of 21 Women During and After Pregnancy



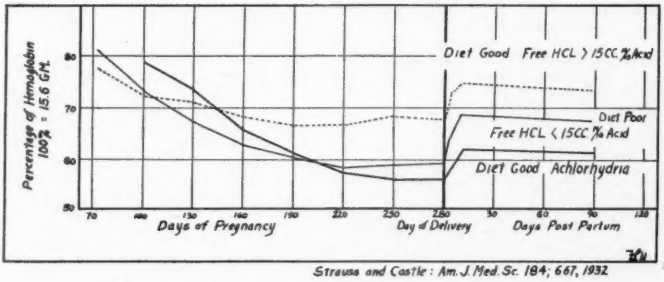
Strauss and Castle: Am. J. Med. Sc. 184, 660, 1932

Lowering of gastric hydrochloric acid in pregnancy has also been reported by Artz[15]. It seems pertinent that Strauss' composite curve for gastric hydrochloric acid in pregnancy and puerperium follows the hemoglobin and cell concentration curves of normal pregnancy. It appears therefore that the expectant mother is placed under hematopoietic stress because of the parasitic action of the fetus, a tendency to defective gastric secretory activity, and inadequate ingestion of hemapoietic substances. The relation of hemoglobin concentration to diet and gastric secretory activity in pregnant women is shown in Chart III. The iron balance studies of Coons[11] indicate however

that even though the gravida has a poor iron-intake, the element is conserved by significant positive balances. Many cases however fail to retain sufficient quantity to meet their own and fetal demands. It appears then that hypochromic anemia occurs in women who have gastric secretory defects, inadequate diets or both. They show progressive decline in hemoglobin and cell concentration which reaches its maximum in the last weeks of pregnancy. They have weakness, pallor, easy fatigue, brittle nails, dysphagia and, in severe cases, dyspnea and edema. The anemia is infrequently as severe and the symptoms as exaggerated as in the macrocytic anemia of pregnancy. The blood picture clinches the diagnosis. The red blood cells ranges from 55 to 75 cubic microns in contrast to a normal of hemoglobin (iron deficiency) than of the number of cells with a resultant low color index (0.4 to 0.7). In the average case the hemoglobin is 6 to 8 Gm. per 100 c.c. (40 to 50 per cent). The average amount of hemoglobin per individual cell is 15 to 20 micromilligrams (normal 27 to 31).

Chart 3

Arithmetic Average Hemoglobin Curves of 17 Pregnant Women Grouped According to Diet Taken and Amount of Gastric Hydrochloric Acid



Strauss and Castle: Am. J. Med. Sc. 184; 667, 1932

The red cell volume is reduced. The mean volume of individual cells ranges from 55 to 75 cubic microns in contrast to a normal of 82 to 92. There is therefore a microcytosis as well as hypochromia.

There is no evidence of blood destruction, hyperbilirubinemia. The leukocytes are numerically and differentially normal. The platelets are not reduced. Anisocytosis and poikilocytosis are common.

The course of the disease is chronic and progressive and accounts frequently for the lassitude and easy fatigue of pregnant women. It has a tendency to subside after pregnancy (probably not uniformly) and to recur in future pregnancies.

Iron, the etiologic deficiency, is a specific therapeutic agent. Large doses are uniformly recommended, 6-8 Gm. of ferric ammonium citrate, or .6 to 1 Gm. of ferrous sulfate daily[15]. Except in asso-

ciation with macrocytic anemia it is unaffected by liver extract or Castles' intrinsic factor.

Hyperchromic Macrocytic Anemia

This syndrome is considered a rarity, perhaps to some extent because of a general lack of understanding of the nature of the disease. Studdiford[16] quotes Gupta that it occurs only once in 10,000 pregnancies in temperate zones. Rowland[17] observed it in 6 of 28 and Strauss[9] in 10 of 40 severe anemias of pregnancy. Indian macrocytic anemia is said to occur in 2.39 per cent of hospital deliveries[20]. I have observed it four times in about 9,000 deliveries.

Since sprue, pellagra and beri-beri are so closely allied to the syndrome and so common in our locality, the incidence is probably greater in the South than is generally appreciated. Unrecognized and untreated, it has a maternal mortality of 65 per cent and a fetal mortality of 40 per cent.[20]

Many theories have been advanced as to the etiology of this syndrome. A specific toxin, syncitial, hemolysin and bone marrow insufficiency, with the ingrafted stress of pregnancy are some of the older theories concerning its cause[21]. Experiments conducted by Castle[23] on patients suffering from pernicious anemia in relapse showed the following: When they were fed ordinary beef muscle with or without acid there was no evidence of increased blood production. Likewise when given gastric juice from normal individuals there was no evidence of increased hematogenesis. When given beef muscle and normal gastric contents at pH 7, prompt blood generation occurred as evidenced by reticulocyte increase, mounting red cells and hemoglobin concentration. Iron given both in combination with hydrochloric acid and normal gastric secretions produced minimal and imperfect hemapoietic response. These experiments gave rise to the Castle theory of the etiology of pernicious anemia, i. e., there is present in the stomach a substance, the intrinsic factor, neither hydrochloric acid nor the commonly known ferments, that reacts with an extrinsic factor present in beef protein and certain other foods which is necessary to maturation of red cells. In its absence macrocytic anemia results. Castle suggests then three mechanisms whereby the deficiency of pernicious anemia occurs:

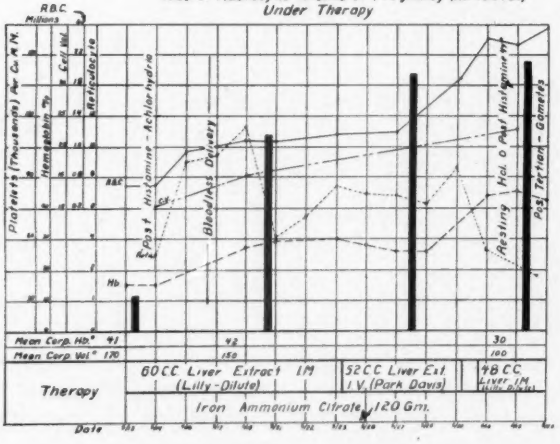
1. A lack of specific intrinsic factor.
2. A chronic lack of extrinsic factor because of poor diet.
3. A failure of absorption and utilization of the product of reaction between the intrinsic and extrinsic factor.

Similar experimental observations[23] were carried out on women suffering from macrocytic anemia of pregnancy before and after parturition. These observations lead to similar conclusions, i. e.,

"The macrocytic anemia of pregnancy, like other anemias responding to liver extract, is due to a deficiency state brought about either by lack of extrinsic factor in the diet, a lack of intrinsic factor in the gastric juice or a combination of the two. . . . In addition a concomitant state (iron deficiency) corresponding to that observed in hypochromic anemia of pregnancy, is frequently present." The uniform absence of gastric hydrochloric acid as is seen in Addisonian anemia was not observed in the pregnancy cases. Recent observations of macrocytic anemia secondary to hepatic diseases as cirrhosis[22] suggest that possibly hepatic insufficiency may be a causal factor in some of the pregnancy cases (Hoffbauer's "Liver of Pregnancy").

The syndrome differs in only a few respects from ordinary Addisonian anemia, i. e., it is limited to pregnancy; it disappears after delivery, occasionally recurs in future pregnancies, does not have uniform post-histamine achlorhydria; fever is frequently present without evidence of infection; neurologic involvement is infrequently observed, and the patients seem to require more massive doses of liver extract than ordinary pernicious anemia patients in relapse.

Chart 4
Changes in Blood Picture and Gastric Hcl
of
Case of Macrocytic Anemia of Pregnancy (No 266781)
Under Therapy



* In Cubic Microns
* Micro Micro Grams Per Cu M M

The examination of the blood shows[25] marked reduction of erythrocytes which is more pronounced than the hemoglobin loss so that the color index is above 1. Anisocytosis, poikilocytosis, polychromatophilia are common. The mean corpuscular volume is increased (105-130 cubic microns). The concentration of cellular hemoglobin is frequently reduced though usually elevated. The leukocytes and

platelets are either slightly reduced or normal with occasional relative lymphocytosis. Icterus index is either normal or slightly increased.

These patients respond to liver extract. From the standpoint of efficiency and economy parenteral liver is preferable (5 to 10 c.c. liver extract N. N. R. daily).

The following case from Shreveport Charity Hospital exemplifies the common characteristics of the disease and shows certain unusual features:

Report of Case

Mrs. E. W., 21 years old, in her third pregnancy, was admitted to the obstetric service on May 12, 1935. Her first pregnancy had been uneventful. During the second she had developed weakness, lassitude, pallor, and some dyspnea in the last trimester. She had been told she was toxic. She had been delivered of her second baby by version and extraction because of a shoulder presentation. Her past history was otherwise irrelevant and she had no family history of anemia. She did not remember the date of her last menstrual period, but reckoned she was at or near term.

She had progressed well during this pregnancy until eight weeks before admission to the hospital. At that time she had developed a severe diarrhea of about 15 bulky foamy stools a day, along with rapidly progressive weakness and dyspnea. She did not know which symptoms had come first. At the time of admission she had been bedridden for three weeks and the dyspnea had progressed to orthopnea. For the previous two months her feet had been swollen and had tingled. Her mouth had been so sore she could hardly eat.

She gave a history of fever of four weeks duration and had been given quinine on May 9 and 10 with no relief. Three days before admission she had broken out with spots all over and blurred vision developed. Her diet had consisted of fresh vegetables, biscuits, syrup, rice and pork. She had not taken cereals or beef.

She was a rather obese blonde woman apparently 40 years old propped up in bed gasping for breath. Her skin showed a waxy pallor and the entire body surface was spotted with petechiae, some of which were as large as a nickel. Her pulse was rapid and weak. The blood pressure was 130/60. Oral sepsis was extensive and the tongue was bald and red. The conjunctivae were icteric and the eyegrounds presented multiple fresh hemorrhages. There was an untransmitted systolic basal murmur. The lungs showed many congestive rales with no evidence of consolidation.

The pregnancy appeared to be near term and the fetus presented the breech at the inlet.

The deep tendon reflexes were normal. Vibratory sense though intact was interpreted to be slightly impaired.

The Kahn was negative. The urine showed traces of acetone and albumin, a low specific gravity, many casts and pus cells in clumps. A stool specimen was negative for occult blood, ova, cysts and parasites. Non-protein blood nitrogen was normal. Icterus index and bleeding time were not determined. A catheterized specimen of urine on admission showed many pus cells.

Red cells, hemoglobin and thrombocyte pictures on several dates are shown in Chart 4. From May 13 to 19 her white cell count varied from 25.2 to 33.1 thousand with 82 per cent and 96 per cent of neutrophils and 1.5 per cent to 3 per cent myelocytes. On June 15 she had 6.9 thousand leukocytes with 60 per cent and 34 per cent of neutrophils and lymphocytes respectively. Though blood cells were examined repeatedly, malarial parasites were not found until June 20.

Her clinical course was tedious though satisfactory. No new petechiae were seen after two days of liver therapy. She professed marked subjective improvement daily and the diarrhea cleared promptly without supplementary medication. Her temperature varied from 103 to 99 degrees till her fourth day postpartum when it underwent incomplete lysis. On June 17 and 19 she had high fever.

Though still very ill she was improved at the time of delivery on May 15. Labor was short (4 hours) and delivery significantly bloodless. The baby weighed 5 pounds and 4 ounces. On its fifth day of life after the onset of icterus it had 80 per cent hemoglobin and 4.74 millions red cells per cubic millimeter.

The patient left the hospital on June 20 with a letter to her family physician for continued treatment. Unfortunately we have been unable to follow her further progress.

Discussion

It is notable that the baby did not share the maternal anemia. This is the rule in all anemias of pregnancy[8,24]. However, they do develop anemia early in life while on a milk diet, a poor source of iron. This is attributed to an inability of the fetus to acquire a sufficient storage of hematopoietic substance in its liver during intrauterine life to tide it over the nursing period.

On admission the patient had 580,000 red cells per cubic millimeter and a hematocrit of 16 per cent. It is noted that the mean volume of her red cells was nearly twice the normal of 82 to 100 cubic microns. The hemoglobin was 16 per cent, or 2.4 Gm. per 100 c.c. Administrations of 10 c.c. of liver extract N. N. R. produced a promptly mounting blood count and reticulocytosis. The latter, in spite of massive doses of liver, was not as great as is usually seen in the severe grades of pernicious anemia in which the degree of reticulocytosis is inversely proportional to the cell paucity. In a week, however, she produced half a million erythrocytes, doubling her cell quantity. In the same period the size of the red cells reduced, as is shown by the cell volume and by the drop in mean corpuscular volume. Declining cell size and cell hemoglobin concentration under liver therapy as shown in this patient is also seen in pernicious anemia in relapse under the same treatment.

In Addisonian anemia secondary reticulocyte peaks are seen when adequate liver therapy immediately follows inadequate amounts. The delayed crescendo in red cell production starting on May 27

may have been due either to the cumulative effect of hematopoietic substances, a beginning recovery from the effects of pregnancy, or a change to intravenous liver.

On May 31 she was transferred to the medical service where she received smaller doses of liver, probably inadequate. The hemoglobin followed the red cell curve satisfactorily with supplementary iron medication, as has already been mentioned. In addition to a deficiency correctible by liver, these patients have a deficiency correctible by iron. The return of gastric secretory function under liver therapy as exemplified by this case is the rule in macrocytic anemia of pregnancy, and is at least indicative of return of the intrinsic factor.

The purpuric manifestations are of particular interest. Normally the blood contains 300,000 to 800,000 platelets per cubic millimeter, and when reduced to below 100,000 spontaneous hemorrhage occurs[27]. Reduction of blood platelets is part of the usual picture of pernicious and pregnancy macrocytic anemia. Though platelet reduction to the bleeding level may have been described in the pregnancy anemia, I have not had access to such literature.

In a very complete review of the subject of purpura with pregnancy, Rushmore[28] finds the complications to be indeed serious. Of 44 mothers with the complication, 26 died. Half the babies were lost. Premature labor was common. Though our patients presented a profound thrombocytopenia a few days before delivery the platelets had probably risen above the bleeding level at the time of parturition.

Why did this patient present this extreme thrombocytopenia and why did the platelet concentration improve under therapy? Secondary purpura is produced by a multiplicity of factors. In the case at hand, avitaminosis and focal infection are possible etiologic factors. Her dietary history does not suggest the former and the latter is untenable in the face of the more apparent explanation; though the physiology of bone marrow is not well understood it is assumed that a common stem cell produces megaloblasts, the precursor of erythrocytes: myeloblasts, the precursor of granulocytes, and the megakaryocytes which produce platelets[27]. This megakaryocyte is reduced in the bone marrow of macrocytic anemia. This suggests that the same deficiency of maturing factor delaying normoblastic activity might in its absence cause an inhibition of platelet formation. If this were true, then liver extract should be specific for idiopathic purpura. Our friends the internists tell us this is not true. Perhaps, then, the megakaryocyte is only relatively reduced in bone marrow incident to megaloblastic hyperplasia.

The arterial capillaries of bone marrow do not anastamose but open directly into the collecting sinusoids. It seems reasonable, therefore, that the presence of large numbers of megaloblasts in a rigid casement of bone might by pressure mechanically interfere with delivery of platelets to the general circulation. The maturation of megaloblasts incident to liver therapy relieving pressure probably permits the platelets to gain access to the general circulation.

The delivery of leukocytes to the circulation is by ameboid activity, and is augmented by chemotaxis incident to infection, etc. This phenomenon suggests that in spite of mechanical impediment leukocytes were delivered to our patient's circulation adequately because of the accompanying pyelitis.

Unfortunately, I have had no direct access to Willis' work on the so called Bombay anemias, similar in every respect to the case at hand. It is said that they are frequently associated with intestinal parasites and malaria and respond well to marmite without elimination of the infestations.

The malaria was not discovered in our case until the patient had more or less recovered. This was because it had been rendered latent by quinine, she acquired the infection while in the hospital, or the plasmodia simply had not been found before. Castle[30] has observed that malaria in anemic bloods is frequently not found until the anemia is corrected.

It is perhaps of some interest that our patient had a history of previous toxemia and evidence of toxemia in the pregnancy described. In this connection it is pertinent that Strauss has observed that late pregnancy toxicoses improve when given high protein diets. His findings of hypoproteinemia suggest that dietary deficiency similar to that producing pernicious-like anemia might also predispose to late toxemia. Perhaps this accounts in some measure for the frequent association of anemia with toxemias, and the liability of toxic patients to shock and infections.

The proper management of pregnancy anemia is, of course, prophylactic, which calls for improving our standards of prenatal care. A satisfactory plan would necessitate erythrocyte and hemoglobin determinations once in each of the first two trimesters and monthly in the last period of pregnancy. There already exists a tendency to make apothecary dumping grounds of prenatal patients, and I shall therefore not suggest routine prophylactic hematopoietic medication.

The curative measure in the several kinds of these anemias has already been discussed. The physiologic type is probably normal. The hypochromic type does not endanger life except by rendering the patient liable to complications.

Liver as a specific agent, preferably parenterally, for the macrocytic type has been mentioned and its efficiency along with that of iron is illustrated by this case. We have record of a similar case in Charity Hospital that died on oral liver because of poor absorption and utilization. Interruption of pregnancy is not indicated. These cases notoriously take a downhill course after delivery. This was demonstrated by the case just referred to. Transfusion takes a secondary therapeutic role. Liver is more efficacious. Transfusions are not without danger in such desperately ill patients. Future pregnancies are not contraindicated. The syndrome has a somewhat doubtful tendency to recur, and adequate supervision and therapy will protect them.

Those of us who are maternal-mortality conscious should take cognizance of the role of anemia in maternal deaths. It undoubtedly contributes both primarily and secondarily to our rather deplorable mortality in Louisiana. In reviewing the situation in Louisiana, I found "pernicious anemia" on 2 of 343 death certificates signed by Louisiana physicians in 1934. Because of the frequent association of anemia with toxemia, these deaths are probably frequently assigned to the classification of "other toxemias." The subject of anemia is, then, highly important in Southern States. Let us give it its deserved attention.

SUMMARY

A brief review of recent American literature on the obscure anemias with pregnancy as a more or less disposing cause is given.

A case of severe grade macrocytic anemia of pregnancy, complicated by purpura, malaria and pyelitis, is reported with discussion of certain hematologic and clinical aspects.

Special reference is made to the purpuric complication, an unusual and dangerous complication of pregnancy. Pregnancy anemia is compared with primary anemias.

It is suggested that the increased plasma volume noted in pregnancy may be due to a compensatory mechanism necessitated by the interposition of the placental circulation which is in effect an arteriovenous fistula.

552 Merrick Street.

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SOME PRESENT DAY CONCEPTS OF BREAST TUMORS

JULIAN L. RAWLS, M. D.

Norfolk, Va.

OUR concepts of pathologic processes in the breast are necessarily changing. We are seeing a larger number of the earlier manifestations of breast disease due chiefly to the dissemination of cancer information to the general public on the part of Bloodgood and the American Society for the Control of Cancer. Bloodgood made the statement several years ago, before all this propaganda was started, that of all women who came to him with tumors in the breast, 85 per cent showed malignant change. Shortly before his death he stated that 85 per cent were then benign. This, of course, gives pathologists an opportunity of studying these various tumors in their earlier stages and of watching them pass out of the benign column into the malignant, and it has changed our thinking of some of these breast conditions.

In speaking of desquamative epithelial hyperplasia or mazoplasia and cystiphorous desquamative epithelial hyperplasia, chronic cystic mastitis or Schimmelbusch's disease, I wish to acknowledge my indebtedness to the recent work of Sir Lenthal Cheatle and Max Cutler[1]. In some instances I have accepted their nomenclature, while in others I have attempted to give their ideas without actually quoting them verbatim. I think it is generally acknowledged that mazoplasia is not a precancerous condition: that it is a normal physiologic process which may occur in male and female breasts alike and that it normally does occur in female breasts at birth, puberty and during lactation. Geschickter, Lewis and Hartman[2] have reported 95 cases of gynecomastia in the male breast and 25 cases of virginal hypertrophy associated with this condition and think it is definitely related to the estrin hormone. They believe "the etiological factor is an abnormally high concentration of oestrin in the circulation, acting upon apparently normal breast tissue."

I have stated that this is not a precancerous lesion. However, it is apparently one of the causative factors in the development of fibro-adenomas, and a small proportion of fibro-adenomas in time will undergo sarcomatous change. The symptoms of mazoplasia are, according to Cheatle and Cutler, diffuse pain, increased density in the gland and a fine nodularity occurring more often in both breasts. It is worse at the menstrual period and does not as a rule develop into true cyst formation. The pain is increased by jarring and sometimes by emotional upsets. Cutler thinks that he has obtained some

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relief by the administration of ovarian residue, probably to offset the excess of estrin. Geschickter, Lewis and Hartman believe they have obtained some results from injections of the milk-producing hormone, prolactin.

Chronic cystic mastitis, Schimmelbusch's disease, is an entirely different pathologic process with a very different prognosis. Cheatle believes it is responsible for at least 20 per cent of breast carcinomas, but states that this process may go on for thirty years before carcinoma develops. It passes through a regular series of pathologic changes, first a desquamative stage in which the ducts and acini may be filled with epithelial cells. As this stage progresses cysts may form in the ducts or acini. The age instance of this stage is the late twenties and early thirties. In the second stage, beginning in the late thirties and early forties the desquamative process has changed from benign epithelial cells to cells that appear definitely neoplastic, although they are still confined within the normal boundaries of the ducts and acini. Single or multiple papillomas are common in this stage. In the third stage, which begins in the late forties and early fifties, the benign condition passes into malignancy in which the neoplasia has broken through the normal boundaries and is then definitely invasive. Secondary deposits may be found in the breast and lymphatic involvement of either the lymphatic channels or the lymph nodes of the axilla or elsewhere may be present. We have to remember that it may be possible to have malignant invasion of the lymphatic channels without carcinoma of the lymph glands themselves. Sections of the lymphatic channels high in the axilla may show involvement without actual metastasis in the glands. Of course if this condition were known preoperatively the patient would be placed in the inoperable class. The question of operation in Schimmelbusch's disease is a serious problem. Certainly the excision of a single cyst offers the patient very little chance of cure, and may be actually dangerous, since the cysts are usually multiple; and, if one single benign papilloma is found, it does not mean that there may not be multiple papillomas scattered elsewhere in the breast, some of which may already be malignant. By taking a small section of the breast and receiving a non-malignant pathologic report, we are given a false sense of security which may end disastrously for the patient. Cheatle is convinced that if we attempt any type of operative procedure, at least half of the breast should be removed, and that probably a simple mastectomy would be preferable. Then the entire breast should be subjected to minute pathologic study, and if a malignant focus is discovered, radical amputation should be done.

We have then occurring in the breast two separate conditions which produce nodularity and discomfort. One is a benign hyper-

plasia seen in young women at or shortly after the time of puberty, and in slightly older women who have not married or who have married but have not borne children. It is the most common cause of adenoma-fibroma before the age of 25. It is very resistant to treatment and does not produce carcinoma. The other is seen in slightly older women; the irregularities in the breast are larger and more pronounced, it runs through a course in which definite changes occur over many years and may eventually be responsible for 20 per cent of carcinomas of the breast. We must remember, however, that the two conditions may occur in the same breast at the same time.

A few words are in order about some of the factors that may play a part in the production of malignancy and may be of some benefit to us in the treatment after malignancy has been diagnosed. We have already mentioned the fact that certain of the female sex hormones, particularly those of the estrin group, play a definite role in the development of the benign hyperplasias of the breast. It is also true that they constitute a factor in the malignancies. There is developing a more definite belief that heredity is one of the primary causes of malignancy anywhere and that, ingrafted on this hereditary tendency, chronic irritation plus certain endocrine imbalances may be the combination of circumstances which are responsible for cancer. There seems to be some definite relationship between the cancer producing elements in tar and in estrin. Loeb[3] states, "Such carcinogenic action is restricted to the tissues of the mammary glands, with which they combine and in which under normal conditions they produce growth processes." In other words, estrin rubbed on the skin of a mouse susceptible to cancer does not produce cancer of the skin, but apparently activates and increases the tendency to cancer of the breast. This naturally brings up the question of the safety of using estrin producing hormones for a long period of time to combat menopausal symptoms, either naturally or artificially produced, in women who have an hereditary history of carcinoma. It has already been claimed that if women within the child-bearing period who have had one breast removed for cancer should develop a subsequent pregnancy, they are very prone to cancer in the other breast. Several years ago Trout[4] collected 15 such cases, 13 of whom developed cancer in the remaining breast. We have long known that there is some activating influence on malignancy produced by pregnancy and that the woman with cancer anywhere in her body who becomes pregnant is frequently doomed to an early death.

We have also to consider the question of roentgenologic castration of women who have cancer of the breast. There is a diversity of opinion on this. Several cases have been reported in which distant

bone metastases from breast malignancies have improved after artificially induced menopause. Recently I asked in one of the larger clinics if they routinely induced a menopause in cancer of the breast, and the reply was, "Not often, certainly not in young women." Apparently then they were not benefiting the women who stood the greatest opportunity of benefit by castration. The older women probably did not need it. Realizing the fact that 96 per cent of carcinomas of the cervix are of the epidermoid type and consequently have a reasonable amount of radio-sensitivity, I think we should not lose sight of the fact that all of these women are sterilized in the course of their treatment, and possibly some of the benefits obtained may be due to the fact that we have entirely destroyed ovarian function. I believe that women developing cancer of the breast probably should be sterilized as a precautionary measure, particularly if they are still within the child-bearing period, and that the menopause symptoms so produced should not be controlled by estrin.

No discussion of tumors of the breast would be complete without a few remarks on Paget's disease. There is a difference of opinion whether to classify it as a benign or malignant lesion. Cheatle describes it as "a slowly spreading, rosy red, dry rash covered by small dry scales having a hard defined abrupt edge and affecting only one nipple." From the lesion Paget's cells can usually be recovered. It may exist for a number of years, spreading slowly, gradually destroying the breast concentrically from the nipple. It does not metastasize and axillary involvement is never seen. Similar lesions have been seen in other locations on the body, spreading slowly and also containing Paget's cells. As the disease progresses in the breast, true duct carcinoma is almost certain to develop beneath the superficial lesion and there has been considerable argument as to which really appeared first. Some claim the nipple lesion is only the end result of irritation from the carcinoma beneath it. Opposed to this theory is the claim that duct carcinoma appearing elsewhere in the breast and invading the skin does not present the picture of Paget's disease. Certainly when axillary involvement occurs the deep seated carcinoma is already present. I consider Paget's disease sufficiently malignant to demand simple mastectomy with immediate examination of the breast by a competent pathologist. If lesions are found elsewhere in the breast, then radical amputation is at once indicated. Cohn[5] recently reported five cases of Paget's disease of the nipple. In one the ulcerated nipple was removed and showed cancer cells. Immediate radical amputation was done and an axillary gland showed carcinoma. No other tumor was found in the breast. He feels that ulcerations of the nipples which

do not respond to treatment in three or four weeks should be widely excised and subjected to biopsy. If carcinoma is discovered radical operation is imperative.

The role played by irradiation in the treatment of carcinoma of the breast is a question of much controversy. At the present time the radio-sensitivity of a tumor is the prime consideration and, unfortunately, pathologic grading does not always answer that question. Presumably a tumor with a grade 3 or 4 malignancy (Broders' classification) should be highly sensitive and the lower grades of malignancy more radio-resistant. This is not always true. The radiologists tell us they have no way of determining this without first subjecting the growth to a course of deep x-ray therapy and then watching the rapidity of its regression.

Coutard states that about 20 per cent of tumors of the breast are very radio-sensitive and that they should be treated by irradiation alone. In this group he places those tumors which have appeared suddenly, and shortly after being noticed by the patient, there developed axillary involvement. He claims that a large number of these patients will respond to a rapidly accumulating dose of deep x-ray therapy and that practically all of them will die of malignancy if they are operated upon either before or after irradiation. His second group embraces 60 per cent of breast tumors and includes those which have grown rather slowly, are quite hard and in which axillary involvement developed rather late. He believes that these should be subjected first to a full course of deep x-ray therapy, rapidly developed, and then at the opportune time, the twentieth to the twenty-fifth day subsequently, should be subjected to radical amputation. The third group comprising the remaining 20 per cent, shows practically no response to irradiation and, if this were known beforehand, should be treated by surgery alone. This group consists of those slowly growing tumors of the breast, of which the woman has been aware for a number of months or years, and which, at the time of observation, are without axillary involvement.

Coutard's treatment is preoperative irradiation. He believes there is a periodicity in the response of both normal and tumor cells to irradiation and that this response shows itself on definite days in epithelial cells and that this number of days depends upon whether the tissue is in mucous-lined surfaces or on the skin. For instance, it appears on the thirteenth or fourteenth day in mucous membranes and on the twenty-sixth to the twenty-eighth day on the skin, so he plans his treatment to deliver the required dose to the particular cells in question on the day of their greatest response. He further states that irradiation, to be of any benefit, must be delivered before surgery is instituted. He states very definitely that

he does not give postoperative irradiation except where known areas of cancer have been left behind. He believes it would be utterly impossible to give a full destructive dose to all the areas where secondary deposits might develop without serious injury to the patient; that if he gave an inadequate dose and cancer later developed, he would have produced, as a result of such inadequate irradiation, a radio-resistant cell which would not respond to treatment. He prefers to wait until the development of secondary deposits, if they should appear, and then give them a full lethal dose.

It would seem, then, that the highly undifferentiated and anaplastic tumor appearing suddenly and growing rapidly belongs to the radiologist alone. Practically none of these will survive five years when treated by surgery. The more differentiated tumor belongs both to the radiologist and surgeon in the order named. The highly differentiated, slowly growing tumor belongs to the surgeon alone.

Recently the external application of large radium bombs or the interstitial implantation of radium salts in properly screened needles after the method described by Keynes of England and Henry of Canada, or the use of deep x-ray therapy by the present high voltage machine, have been advocated. Henry apparently prefers to treat all types of breast tumors by interstitial irradiation. The time has been too short to evaluate these methods of treatment. However, it seems they have nothing to offer in the way of irradiation that cannot be accomplished by any modern deep x-ray therapy machine in the hands of a competent radiologist. There is no difference in the effect of properly delivered gamma rays whether they come from properly screened radium or x-ray. It is true that the set-up of the x-ray machine is less expensive and that the treatment is less hazardous to the patient.

Report of Cases

Case 1. A white matron, aged 48, had first noticed a small mass about the size of a large marble beneath the skin of the right breast three weeks before admission. This was not tender nor painful and gave no symptoms at all. As it continued to grow larger she consulted a physician and after his examination she noticed a slight tenderness and occasional aching in the right breast. The mass had grown to the size of a hen's egg. She had noticed no other masses in proximity to the breast. She had lost about 20 pounds in the past two or three months but thought this loss due to an anti-obesity diet. There was no history of injury to the breast. Her general health had been excellent and she had never had a serious illness or operation. She had had one pregnancy 25 years before and had nursed her baby fifteen months.

Physical examination revealed nothing unusual except a blood pressure of 170 systolic and 110 diastolic and the condition of her right breast. Both breasts were large and pendulous. The right breast contained, on the medial side, a mass the size of a small orange, attached to the deep structures but not

to the skin. The skin over this area was slightly hyperemic and warm, but showed no dimpling or ulceration. Her temperature was 98.8; pulse 80; white cells 10,500 with 73 per cent neutrophils. White count six days before was 10,400 with 80 per cent neutrophils.

Roentgenologic report: "Right lung: There is a dense rounded shadow, approximately 1 cm. in diameter, toward the upper portion of the lung field, which is probably a metastatic lesion. The lower trunks show some peritruncal hyperplasia. The upper and peripheral portion of the lung field appear clear. Left lung negative. Conclusion: Radiographically the findings are indicative of metastatic lesions of the right lung. There is a possibility that this may be a calcified healed tuberculosis lesion but this is doubted."

The following day a radical breast amputation was done, using a radio-knife. Several enlarged axillary glands were encountered. The following is the laboratory report: "The specimen consists of a large breast containing two abscesses with streaks of fibrous tissue around them. Microscopic examination: One section shows typical abscess formation. The cavity is lined with necrotic tissue in which there is a heavy infiltration of neutrophils and large mononuclears. This infiltration is most pronounced about the resting milk glands. There is no evidence of malignancy. Axillary gland: The section shows congestion, edema and a slight infiltration of neutrophils and large mononuclears. Diagnosis: Abscesses of the breast and subacute lymphnoditis."

She was re-admitted to the hospital 14 months later in profound shock from apparently some acute abdominal catastrophe and died within a few hours. No autopsy was obtained.

CASE 2. A white matron, aged 29, was first seen January, 1931, when she was about 8 months pregnant. She presented herself because of a hard, fixed mass in her left breast which was on the point of ulcerating through the skin. She gave this remarkable history: She had had six previous pregnancies and in either the first or second she had an abscess of the left breast which had been opened and drained. The lump in that breast had never disappeared but she had nursed her babies following each of her subsequent pregnancies from both breasts alike. There had been no further infection in that breast. She sought medical advice because the mass had grown and had become painful.

X-ray showed marked increase in density and an infiltration at the base of the left lung which extended out to the periphery. There were multiple dense nodular shadows in the lung field to the outer side of the hilus region. The radiographic conclusion was infiltration at the base of the left lung, probably malignant.

The treatment here was a difficult problem. She was eight months pregnant and she unmistakably had a carcinoma of her left breast with probable metastasis in her left lung. Operation, of course, was out of the question and we were afraid that x-ray therapy would very likely produce a miscarriage. The interesting question was just when this mass in her left breast, which had been present through five different lactations, swung over into the malignant column and what had activated its growth? Radium, in needles and tubes, was buried beneath and around the growth, and it promptly regressed. She was delivered a month later and was not allowed to nurse her baby from either breast. Her general health immediately began to improve. She put on weight;

her appetite was good; she had no cough and we began seriously to doubt if our interpretation of her x-ray plates was correct. She was kept under observation until June when she failed to report for a check up.

She returned to the hospital the latter part of November and stated that in August she had developed an acute cold and had been going rapidly down hill ever since. At that time she had some edema of her ankles and was unable to lie flat in bed on account of dyspnea. She had evidence of marked involvement of both lungs, both from physical examination and x-ray findings. She died in December of that year.

In view of the course in this case we cannot help wondering if roentgen sterilization followed by deep x-ray therapy of her chest might not have offered her some prolongation of life.

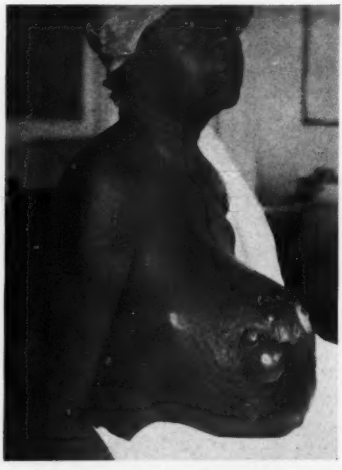

Fig. 1. This negro woman, aged 45, first noticed a small hard lump in the right breast four months before the photograph was taken. (Case 5.)

CASE 3. A white matron, aged 41, reported in October, 1933, with a small hard lump in the upper medial portion of her left breast which she stated she had first noticed three weeks before. It had increased slightly in size and was painful. She had had one pregnancy 13 years before, but no previous illness of any significance. Neither axillary nor supraclavicular glands were palpable. The small mass was not attached to the skin and was movable over the pectoral fascia. A diagnosis of probable fibro-adenoma was made. Biopsy was performed and reported clinically malignant. Immediate radical amputation of the breast was done. The fixed specimen was reported as having some sections that showed a medullary type of carcinoma while others were scirrhous. Two lymph nodes were found in the axillary mass, neither of which showed carcinoma.

She was re-admitted March, 1935, with the complaint that some three weeks before she had noticed a hard fixed gland under her left arm. A diagnosis of recurrent carcinoma was made and, as the mass was fairly discrete and

movable, it was decided to resect it rather than submit her to x-ray therapy. The pathologist reported that the mass was a fibro-lipoma of the axilla with no evidence of malignancy.

There is no evidence of recurrence at this time.

CASE 4. A white matron, aged 28, came to the office February, 1931, because of a small mass in her left breast. She had had one pregnancy 3½ years before and at that time an abscess had developed in the left breast. It had left a small hardened area which varied in size at different times. About 18 months previously she had noticed a small nodule at the outer side of this mass. It was about the size of an almond, hard and freely movable. On examination she presented one small nodule in the left axilla, hard and also freely movable. Although she was advised to have it removed for section, she did not return until May, at which time the mass in her breast was a little larger and there were two small nodules in the axilla. A radical breast amputation

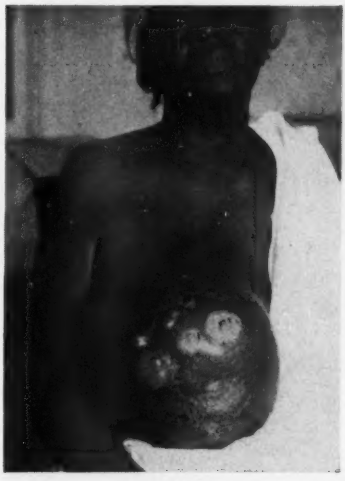

Fig. 2. Another view of the same patient. The pathologic diagnosis was adenocarcinoma of the medullary variety.

was done and the pathologic report was medullary carcinoma of the breast and metastatic carcinoma of the axillary lymph nodes. Immediately following her operation she was given a course of deep x-ray therapy. She was kept under constant observation and in the late summer of 1932 she had palpable supraclavicular lymph nodes. At that time her chest was x-rayed and reported clear for evidence of metastasis. She was given a second course of deep x-ray therapy.

She was re-admitted to the hospital Oct. 26, 1932, 30 days later. At that time she was markedly dyspneic with a rapid pulse. X-ray report: "There is extensive clouding throughout both lungs believed to be due to metastases." Her skin was blue, cold and wet for at least 24 hours before death, which

occurred only five days after admission. Death was essentially from asphyxiation; oxygen inhalations gave her scant relief.

In view of the fact that her chest was reported clear of metastasis on Sept. 12, 1932, and both lungs were entirely filled up by October 21, less than six weeks later, I have always felt that this patient's terminal condition might have been due to the extensive radiation which we gave her.

CASE 5. A negress, aged 45, was first seen July 27, 1936, with the history that four months before she had first noticed a small hard lump in the inner and upper quadrant of her right breast. This had become painful and gradually enlarged until the skin broke down and began to discharge a yellow fluid. She had no loss of weight or strength. No lumps were felt in the axilla at this examination. Within the next week she was given 2,400 R units over the anterior aspect of her breast. It had absolutely no effect on the growth. The breast increased to the size shown in the picture, the axilla became involved and several areas of skin broke down and began to discharge.

The rapidity of the growth made us think that we were probably dealing with sarcoma, and in October, two months later, a simple mastectomy was done to remove the sloughing areas. Pathologic diagnosis: "Adeno-carcinoma of breast (medullary variety) and metastatic carcinoma of axillary lymph nodes."

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708 Medical Arts Building.

SULFANILAMIDE IN UROLOGY

STEPHEN T. BROWN, M. D., F. A. C. S.
Atlanta

SULFANILAMIDE, the name given the new synthetic chemical compound, para-amino-benzene-sulfonamide, has recently received spectacular acclaim from both the lay and medical press. This drug, under the name of prontosil, was first used in 1935 by Domagk, a German chemist, in experiments on mice. Later, he experimented and demonstrated it was bactericidal to streptococcal infections in the human body. During the past few months, it has been shown that this drug is eliminated through the urinary tract. It has also been proved that it is equally effective in treating the bacillary and coccal types of infections of the kidney, prostate and urethra. However, the use of sulfanilamide is as yet entirely in the experimental stage.

The widespread publicity accorded this drug has led the public to believe that gonorrheal infections can be cured with a few doses of sulfanilamide and this has resulted in promiscuous sales by the druggist. This is inviting disaster. While sulfanilamide is, at this time, the most powerful urinary antiseptic known, it is also a treacherous drug demanding extreme care and caution in its administration. It is obvious that a drug with such potent possibilities should have the competent control of the medical profession.

A review of the literature on sulfanilamide reveals publications of extraordinary results obtained by various clinics with the use of this drug in kidney, prostatic and urethral infections. The Mayo Clinic stated that in 24 cases of prostatitis, followed by cultures, only one remained positive when using sulfanilamide; this was in contrast to 50 per cent positive cultures following the use of the mandelic acid. Some clinics maintain that it is effective in all types of infection except that of the streptococcus fecalis. I have not had a chance to treat an infection with this organism.

It has been reported that gonorrhea can be cured in five days. Dr. Edgar G. Ballenger reports that of 26 cases of gonorrhea, 25 were cured with an average of four treatments by the "sealing method" together with the use of sulfanilamide. He sealed the urethra of those patients who reported to him for treatment within the first 24 hours after the discharge appeared. In spite of the reports of so many 5 day cures of gonorrhea, my experience with sulfanilamide in the treatment of 33 cases shows that it requires an average of five weeks to effect a cure. The sulfanilamide was used in addition to the usual recognized treatment for gonorrhea.

My patients applied for treatment when the discharge had been present from three to ten days (average six days). I might add also that some of them had already been taking sulfanilamide on the advice of so-called friends in preference to applying immediately for treatment. However, the time of cure was found to be almost half that needed with the former methods of treatment for gonorrhea.

It is also interesting to note that the use of this drug tends to prevent complications. In the few cases of chronic prostatitis of gonococcal origin, where sulfanilamide was used, I found a marked influence on the termination of the infection. Most of these cases cleared up rapidly, and required only a few prostatic massages before all pus and infection disappeared from the prostatic fluid.

Ballenger contends that, in his experience, sulfanilamide is more effective if a temperature of 103 or more exists. It seems that sulfanilamide itself often produces a fever. If none exists, an artificial fever is produced for a few hours. My results have been the same regardless of whether the patient had a fever or not from the use of the drug.

My results in using sulfanilamide in kidney infections have been astounding and—most gratifying. In one case of proteus infection of the right kidney, all clinical, laboratory and x-ray findings indicated multiple abscesses of the right kidney. The patient was so ill that it was deemed unwise to attempt surgical drainage. Large doses of sulfanilamide were administered and this patient made an amazing recovery. The urine is now free of pus and x-ray pictures made, following the administration of sodium mono-iodomethane sulfonate (skiodan) intravenously, disclosed the kidney had returned to approximately the normal size with good elimination of the dye. There was no evidence of kidney destruction resulting from the infection.

Sulfanilamide tends to become an ideal addition to the present plans of preparing a patient for prostatic resection. It is being used extensively with splendid effect prior to operation. Particularly, it has proved of valuable aid in combating the persistant pyuria that follows these operations. This is in direct contradiction to the previous claims that certain types of prostatic hypertrophy with renal insufficiency were contraindicated to its use. Possibly the explanation is the improvement in renal function by the use of an indwelling catheter for drainage in these cases. At any rate, the sulfanilamide seems to minimize the urinary infection that accompanies obstruction at the outlet or the use of an indwelling catheter.

The initial dose, as recommended, is 80 grains a day for two days. Following this, the dose is reduced to 60 grains for two or more days, depending on the tolerance of the patient. A dose level of 20 to 40 grains a day is then maintained for a period of ten days or until recovery. However, some patients require the small doses in the beginning with a gradual increase as a tolerance of the drug is established. I find that best results are obtained when the maximum dosage is used at the start and that the drug is tolerated better with the patient in bed. The most common complication that we see is sulfhemoglobinemia. It appears advisable to tolerate a moderate amount of cyanosis as there has been a blueness of the lips, gums and nails of practically every patient treated with this drug. Most of my patients have experienced the commonly reported reactions to this drug, namely, malaise, dizziness and partial loss of appetite; there has been a loss of weight in every case. It should be remembered that any condition which will produce renal insufficiency, such as stones, ptosis, stricture of the ureters or urethra, or advanced prostatic hypertrophy, will interfere unfavorably with the action of the drug. If severe headache, gastric symptoms (including nausea and vomiting), diarrhea, chills and fever or dermatitis appear, the drug must be stopped immediately. Extreme care must then be used in returning the patient to the drug. I have one patient in whom one tablet will produce a severe reaction. He was tried on the drug on four different occasions and finally rebelled. In one case, a dermatitis developed similar to that of an arsenical dermatitis. This patient suffered with intense itching wheals and there appeared a redness over the entire body, but the duration of this complication was much shorter than that of an arsenical dermatitis. Two other cases developed a mild dermatitis. However, neither of the three patients would again tolerate the drug without a return of the symptoms. Two patients have had chills occurring 8 to 24 hours apart, and fever ranging from 103 to 105 accompanied by diarrhea. These conditions lasted for a period of five days. Needless to say, further treatment was not tried in these two cases.

A few deaths have been reported from agranulocytosis, presumably from the use of this drug. However, my cases have been followed with blood counts and, to date, have shown an increase in the white blood cells. Apparently, this complication is of prime importance and will necessarily force us to follow cases treated with sulfanilamide with repeated blood examinations.

As this paper goes to press, it is my painful duty to report the return of several patients for treatment of urinary infections. They have returned with the urine loaded with pus and bacteria and are seemingly well on the road to their conditions previous to the use

of sulfanilamide. Apparently, these patients had been cured of their urinary tract infections. Now, after a period of from four to eight weeks without treatment, they have returned with symptoms the same as or similar to those before treatment was instituted. But not so with gonorrhea, for to date I have not seen a recurrence of a gonorrheal infection after a patient was dismissed as cured. So I leave the subject of sulfanilamide, heartily agreeing with Dr. W. F. Braasch who makes the following timely statement: "The therapeutic and physiological limitations of this remarkable compound remain to be determined."

Conclusions

1. Sulfanilamide is the most powerful urinary antiseptic known at this time.

2. Full doses are necessary for best results but such doses sometimes produce serious reactions.

3. The appearance of toxic symptoms requires immediate cessation of the administration of the drug.

4. This drug should only be administered under the very careful and close observation of a physician. A method should be effected for the distribution of this drug, keeping it in the control of the physician who is competent to recognize complications as they occur.

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EPILEPSY

Discussion of the Surgical and Nonsurgical Treatment

R. GLEN SPURLING, M. D.
Louisville

THERE is perhaps no single group of chronic sufferers who are so utterly miserable as those composing the epileptic group. They do not ordinarily die of the disease; many of them must await some intercurrent ailment to relieve them of what has been a horrible existence. Many of them are thwarted in their attempts to make something useful of their lives. Most of them are denied the pleasures of marriage and children because of the hereditary nature of the disease. What may be more pathetic is the social stigma which descends upon the immediate family of the victim. For centuries the sufferers of fits have been spoken of in hushed tones and with lifted eyebrows. They and their families are forced by social customs to be secretive at best and social outcasts as a rule.

That the scourge of the disease has not been eradicated and its treatment made more efficient is not due to lack of interest among clinical and laboratory investigators; scores of brilliant men have devoted their lives to the study of the problem. *Much has been accomplished* and I believe if these accomplishments were better known to the profession, the plight of the epileptic as far as treatment is concerned would be tremendously improved.

I shall attempt to discuss today the practical, rather than the theoretical aspects of the problem. I shall tell you the methods I use in the study and treatment of the various types of epileptics. These methods are not original with me, but they have been subjected to rigorous selection, and many of them modified in an attempt to place them upon a workable basis suitable for the majority of cases.

When the patient enters the hospital—and every case should be hospitalized during the investigation—he is subjected to a complete clinical study. Since convulsive seizures occur as a manifestation of general systemic disease, notably cardiorenal disorders, hyperinsulinism, febrile illnesses of childhood, parasites, arteriosclerosis and chemical poisoning, these causes are eliminated in the beginning. If a primary disease of the central nervous system is present, such as cerebral tumor, cerebral abscess, cerebral birth paralysis, or a traumatic lesion of the brain, a painstaking neurologic examination should ordinarily disclose it. However, it is rather striking how

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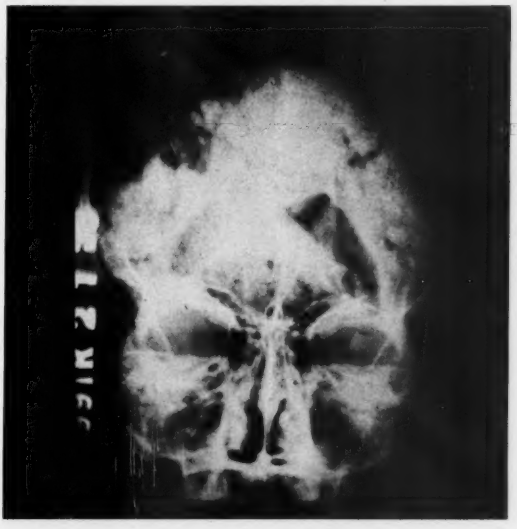
frequently one of these cerebral lesions has been missed clinically in our series of over two hundred epileptic patients, and their presence finally disclosed by air studies—a subject I shall discuss at some length a bit later.

Perhaps the most important part of the clinical investigation is an accurate history of the attacks. This should always be obtained from the patient and some person who has observed him repeatedly. What was the first symptom noted at the beginning of an attack? What did the patient do? How did he act? What was the sequence of events after the onset of the attack? To which side did the head turn? In which direction were the eyes rotated? Did one arm and leg behave differently from the corresponding member? These and many more similar questions and their answers often lead the investigator into an accurate localizing diagnosis. It is often desirable for the physician to observe personally the pattern of a seizure. In most epileptics a convulsion can be induced by hyperventilation (hyperpnea) or by hydration. When an accurate account cannot be obtained, I feel justified in deliberately inducing a seizure.

The most frequent localizing sign is elevation of the eyes and turning of the head to the side opposite the hemisphere involved. Seizures which have their origin in the frontal lobe are usually characterized by loss of consciousness (without aura) and turning of the eyes, head and body to the opposite side. This is followed by a nearly simultaneous convulsion of the opposite extremities, falling, and generalization of the attack. In seizures which arise from lesions just anterior or posterior to the Rolandic fissure, unconsciousness does not ensue until later. A tingling sensation may follow a jacksonian march, just as movement follows in seizures arising in the frontal lobe. In fact, consciousness is apt to be lost late in seizures arising anywhere behind the central sulcus. Such seizures are ushered in by aura. It must be remembered, however, that a major attack may leave retrograde amnesia, so that the aura is forgotten. In such circumstances, the aura may be remembered only in slight seizures which do not progress to generalization. Seizures originating in the occipital pole or in the supramarginal gyrus are characterized by "trembling" or "flashing" of lights, seen in the contralateral field. An aura of epigastric distress or pain is frequently experienced with lesions of the sensory cerebral cortex usually of the postcentral convolution. "Buzzing sounds" and dizziness are characteristic of unilateral temporal lobe lesions. Uncinate attacks, those queer hallucinations of smell, are always pathognomonic of a lesion involving one or both temporal lobes.

With the introduction by Dandy in 1919[2] of pneumo-encephalography, the study of organic cerebral disorders received a tremen-

dous impetus. This important diagnostic procedure has revolutionized the localization of obscure brain lesions and has opened up, because of the additional information obtained, new avenues of treatment. No epileptic has been completely studied until he has been subjected to air studies. Whether air is injected directly into the ventricular system (ventriculography) or into the spinal subarachnoid space (encephalography) is a matter of individual preference. I prefer the latter procedure in those cases without marked



CASE 1.—J. W. H. This first case is a good example of the results which are apt to follow an incomplete primary operation for depressed fracture of the skull. This boy was seen by me in February, 1935. He had his first convulsion four months prior to this date and since the original seizure had had grand mal attacks on the average of three times a week. He gave a history of having sustained a depressed fracture of the skull three and a half years before. He was operated upon in another city and the depressed fracture was elevated. At the time of our examination we found the scalp densely adhered to the underlying structures through the operative defect.

The encephalogram showed a definite cerebral scar with attachment to the meninges and scalp. At operation this scar was verified and removed completely. It extended down into the wall of the ventricle and it was necessary to open the ventricle in order to resect the scar completely. The defect in the skull was repaired with a celluloid plate. The patient made an uneventful convalescence and has been completely well since this date. There has been no recurrence of seizures.

increased intracranial pressure. By replacing the entire volume of cerebrospinal fluid with air, it is possible by means of carefully taken x-ray films to study not only the interior of the brain by visualization of the ventricular system but the exterior of the brain as well by visualization of the cerebral subarachnoid spaces and basal cisterna. Pneumo-encephalography stands in the same relative importance to

neurologic diagnosis as does pyelography to urology and cholecystography to abdominal surgery. Curiously enough, the injection of air into the subarachnoid space has a beneficial effect upon the number and severity of the convulsive attacks. Patients have been known to be free of seizures for many months following this procedure when no other therapy has been employed. However, it is not because of the possible therapeutic benefits that we advise encephalography in every epileptic patient, but because the information obtained provides us with data which may be indispensable for the proper management of the case.

With our knowledge of treatment of epilepsy in its present state, the patients may be divided roughly into two groups: (1) the surgical; (2) the nonsurgical.

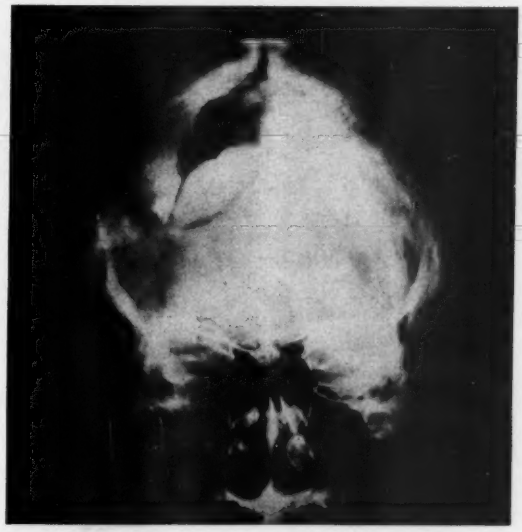
The Surgical

The operative treatment of epilepsy has been the perennial vogue in various clinics for the past forty years. Simple decompression operations, implantation of foreign bodies upon the surface of the brain, various types of cervical sympathectomy, drainage of arachnoidal lakes of fluid, surgical alterations of venous drainage—all of these and many more, such as colectomy, have been employed from time to time with the hopes of bringing relief to the epileptic patient. Needless to say most of these have been lacking in rationale and consequently have been discarded. Today, it is generally conceded that there is no approved or accepted surgical procedure for cases of idiopathic epilepsy. On the other hand, there are two types of epilepsy in which surgery offers very satisfactory end-results: (1) traumatic epilepsy with localized cortical scars, and (2) jacksonian epilepsy with a sharply defined trigger point without scar formation.

The principles laid down by Foerster, Penfield and their pupils[4] form the basis for all modern treatment of traumatic epilepsy. Penfield summarizes the situation perfectly in these words: "If the patient's history, the encephalogram, the pattern of the seizures and perhaps the neurological examination all incriminate the same area of the brain, then electrical exploration is justified. If this exploration is in accordance with the rest of the evidence, complete radical excision of the focal lesion is the rational method of treatment, a treatment which has been justified by its practical results."

In order to carry out satisfactorily an electrical exploration of the brain, it is necessary that the operation be done under local anesthesia or perhaps local supplemented by a light basal anesthesia. If stimulation of the suspected area with a weak faradic current produces a convulsion with the same pattern as observed during the

investigation, whether there be gross evidence of disease or not, then that area should be excised with the electrosurgical knife so widely that further stimulation fails to reproduce the muscular responses already noted. If there be bony defects overlying cortical scars, these are always repaired with either a bone graft or a celluloid plate. I have used the latter method exclusively for the past four or five years with better results and with considerably less work than when massive bone grafts were attempted.



CASE 2.—P. Y. This next case illustrates the drastic methods which may have to be employed to relieve post-traumatic epilepsy. This patient was shot through the right frontal region with a 45 caliber bullet in 1926. The wound in the brain was packed with gauze to control hemorrhage. He developed a brain abscess and osteomyelitis of the skull and after multiple operations the abscess healed. Two years later he began to have convulsions. The convulsions were incompletely controlled with phenobarbital. They have gradually grown more severe and incapacitating until June, 1936, when he reported to the service for operative treatment. It was necessary to resect the whole right frontal lobe in order to remove the scar which was attached to the meninges and scalp through the multiple openings in the skull. He made an uneventful convalescence and has been free of seizures to date. Apparently, his mental faculties were not impaired by this extensive loss of brain tissue necessitated by the radical operation.

While much has been written about the repair of cortical scars in traumatic epilepsy, little has been said about their prevention. In most cases of acute head injury resulting eventually in a localized cortical cicatrix there has been a depressed fracture of the skull with an area of local contusion and laceration to the brain and meninges. It has been a too common practice, if any operation is done at all, simply to elevate or remove the skull fragments and disregard the

devitalized brain tissue. In the process of healing, all such tissue is replaced by an astroglial network, which often becomes thoroughly fixed to the meninges and tissues of the scalp. Such a scar exerts a pull over a widespread area of the brain. If at the time of the acute injury all devitalized cerebral tissue is clearly removed, the resulting gliosis is reduced to a minimum and the cavity thus created becomes filled with cerebrospinal fluid. The likelihood of an extensive scalp-meningo-cerebral scar is thus greatly diminished. A thorough debridement of the entire traumatized area at the time of the acute injury would certainly reduce the incidence of traumatic epilepsy to the minimum.

The Nonsurgical

By far the largest number of epileptic patients (perhaps 80 per cent) fall into the nonsurgical group. They are the ones most difficult to treat chiefly because the etiology is so frequently obscure. After the air studies are completed and the possibility of a surgical lesion has been ruled out, the patient is then started on a routine which in my experience brings some measure of relief to all cases and has enabled others to remain free of seizures over a period of years.

I shall discuss the nonsurgical treatment under four headings:

1. General hygiene.
2. Drug therapy.
3. Ketogenic diet.
4. Dehydration.

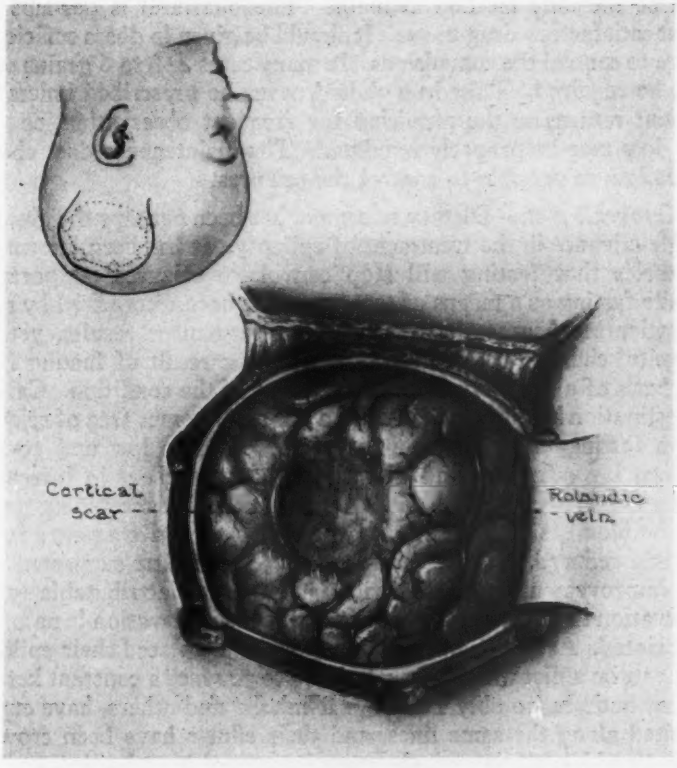
General Hygiene.—It is most important that the patient subject to convulsive seizures should lead a carefully regulated life as free as possible from emotional stress, mental and physical fatigue. A moderate amount of daily exercise in the open air is advisable. Alcohol and all stimulants should be eliminated from the diet. If the patient's occupation is such that attacks endanger himself or co-workers, it should be changed. Likewise, he should not be allowed to operate a motor vehicle or engage in any recreations where, should he have an attack, his own life or that of others would be endangered.

Proper elimination is an essential feature of the routine.

Because of the stigma associated with the term "epilepsy" in the minds of the laity, physicians are reluctant at times to tell the family or patient the true condition. This point of view is wrong. If it has once been established that the patient is suffering from epilepsy, the responsible member of the family should be told. This not only promotes better cooperation from the standpoint of treatment but

avoids having the patient or his family discover the true nature of the malady from sources other than their medical advisers.

Drug Therapy.—For the control of the seizures, drug therapy is by far the simplest method. The bromides have for years been the generally accepted medication for the epileptic. With the introduction in 1912 of phenobarbital (luminal) as a nerve sedative, this



Case 3.—H. M. The last case is an example of what we consider to be a cerebral birth injury resulting in hemiplegia and jacksonian convulsions. While his first seizures did not occur until he was 6 years of age yet he did have a mild right hemiplegia from birth. The attacks have been predominantly focal in nature but progressed rapidly into generalized seizures. In spite of this the boy managed to get through high school and did exceptionally good work. For the previous year, however, attacks had become more frequent and of longer duration. Six months prior to operation he had several attacks each day. With the increase in the number of attacks his mental faculties became clouded and dull. At the time we saw him on July 20, 1935, he was decidedly deteriorated. An encephalogram showed the characteristic deformity of a cortical scar. At operation in July, 1935, this scar was demonstrated and removed cleanly down to and including the wall of the ventricle. Since the operation he has had no semblance of an attack. He again has become bright and alert and is earning a livelihood.

drug has practically replaced the bromides in popular favor. These two drugs, given singly or in combination, do control in a fairly satisfactory manner the number and severity of the seizures. Their beneficial effects are in the majority of instances only temporary, as a tolerance is developed rapidly. There will always be a group of patients in whom, for one reason or another, more exact and desirable methods cannot be instituted. Drug therapy in this group will be the only method available. Phenobarbital is perhaps the most satisfactory drug to use. It should be given in doses sufficiently large to control the convulsions. In many cases 4½ to 6 grains daily will be required. This drug should never be prescribed unless the patient returns to the physician for frequent observation, so that the dose may be properly regulated. The maintenance dose should be as low as possible to control the seizures.

Ketogenic diet.—Dietary treatment has been perhaps the greatest single advance in the treatment of epilepsy. It has been known for centuries that fasting will stop convulsive seizures temporarily. While fasting as a means of treatment has been discredited by most investigators because it does not give permanent results, yet the chemical changes observed in the body as a result of fasting form the basis of all modern dietary treatment of the condition. Careful investigation has shown that when a patient becomes free of seizures from fasting, the sugar content of the blood is low and acetone bodies, i. e., acetone and diacetic acid, are present in increasing quantities in the blood. Also, the carbon dioxide combining power of the blood is diminished and the hydrogen ion concentration is slightly reduced. Wilder[9] and later Peterman[7] first suggested that the improvement associated with fasting is not attributable to the starvation itself but to the ketosis with which starvation is naturally associated. Acting upon this assumption, they placed their epileptic patients on a diet which was calculated to produce a constant ketosis. Many authors, notably Talbot[8], Helmholz[5] and others, have experimented along the same lines, and their efforts have been crowned with considerable success. In many instances, there has been a complete cessation of the attacks over a period of years, and in others, a decrease in the number and severity of seizures. Furthermore, it has been demonstrated beyond doubt that a patient may remain on this diet indefinitely without detrimental effects to the general health. In fact, normal growth and development take place even in the young. Some authors believe that patients maintained on a ketogenic diet are freer from intercurrent illnesses than on a normal diet. This is true particularly in the case of the common cold.

In my experience, it is futile to attempt the diet unless the patient and some other responsible person have been instructed thoroughly

in the practical side of dietetics. Hospitalization for a week or ten days is essential. First, encephalographic studies are made and following this procedure the patient is placed in charge of the dietitian for instructions into the theory and practice of the diet. He is taken to the diet kitchen and taught to prepare his meals under supervision. He learns to test daily his state of ketosis by analysis of his urine for diacetic acid. When he is sufficiently conversant with all the details, he is discharged from the hospital and instructed to report to the physician or to the dietitian when any change is to be made in the diet, or when the diet fails to produce the desired ketonuria.

It is possible for patients in any walk of life to learn this diet and adhere to it rigidly. I have records of laborers who have learned the diet and who have carried it out over a period of years. Young children offer the most difficult problem, because in many instances it is impossible to make them understand the importance of rigid adherence to the routine. Many of them will steal candy or sweets and, of course, spoil the state of ketosis. Other children learn very early the importance of rigid adherence. Dr. Thomas Marks, of Lexington, Kentucky, tells a most illuminating story of one of his little patients who had been on the diet for several months. This boy came into a corner drug store with three of his playmates. The playmates each bought an ice cream cone, and this little fellow showed no inclination to obtain one. A traveling man standing in the store noticed that the boy did not have a cone and feeling sorry for him he suggested that he would be glad to buy one for the child. The boy looked up at him and said: "Thanks, Mister, but I wouldn't eat one of them things for fifty dollars. I would be sure to have a fit tonight if I did."

Dehydration.—The possible relationship between hydration and dehydration to epileptic seizures has attracted the attention of many investigators. Since the days of Hippocrates a moist brain has been thought to be associated with epilepsy. McQuarrie[6] in 1929 showed that there was a tendency for the epileptic to store water during the active stage of the disease in amounts that were harmful. He also showed that convulsions tend to occur when a water balance above a certain magnitude is established, and that after dehydration occurred, convulsions, in many instances, were prevented. He found that it was possible by a sudden increase in the water intake to throw the severe epileptic patient into convulsions, and by dehydrating him to relieve the convulsive manifestations. Temple Fay[3] has been the chief proponent and advocate of this method of treatment. On a basis of his own experience, he finds this method has given relief to patients when other methods have failed.

Dehydration automatically occurs in patients upon the ketogenic diet. Barbour[1] demonstrated that dogs on a high-fat, low-carbohydrate diet take voluntarily approximately 50 per cent less water than animals under similar environmental condition will take on a normal mixed diet. From our experience, we find that patients on a high ketogenic ratio do not desire water in large quantities. I have for several years limited the fluid intake of epileptic patients in connection with the ketogenic diet and have found that they remain comfortable on a fluid intake as low as 600 c.c. per 24 hours. It is my belief that the combination of ketosis plus dehydration is the method of choice in the treatment of the epileptic.

In closing, let me say that for one to appreciate fully the change that has occurred in the attitude toward the treatment of epilepsy, he must read the literature of a decade ago and compare it with that of recent date. More exact information has taken the place of surmises, conjectures and pseudo-knowledge. I do not mean to imply that the problem is solved; it is far from that state, but I have attempted to show that sufficient scientific advances have been made to justify our substituting a spirit of hope and expectancy for that of utter despair when dealing with the epileptic patient.

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THE AMERICAN BOARD OF SURGERY, I

Dr. J. W. D. Dicks, of Natchez, unable to present his paper at the last Assembly of The Southeastern Surgical Congress, allowed it to be used as an editorial in the October SURGEON. The theme of his essay was so forcibly expressed that it must be repeated:

> Too many operators are neither mentally nor morally prepared to undertake the great responsibility of a surgeon and they are doing too many needless operations. The unsuspecting public . . . thinks that the possession of the M. D. degree is prima facie evidence of ability to practice surgery in a capable manner. . . . The license to practice medicine should not confer the right to do major surgery.

To meet this need for a board to certify the professional fitness of surgeons, the organization of such a board under the aegis of the American Surgical Association had already been undertaken: it has held its first examinations since Col. Dicks' paper appeared.

The American Board of Surgery promises to do more to elevate the standards of surgery in these United States than any other factor that has ever influenced them. It also promises to be a powerful influence for the good of all surgical patients.

The Board in its first years may be criticized by some individuals. This should not worry its supporters for even the Supreme Court has been under fire these past ten months. THE SOUTHERN SURGEON earnestly commends the Board to the Fellows of The Southeastern Surgical Congress; it notes with pleasure that several of them took the examinations in October and it hopes that in the near future all the rest will exert themselves to secure its certificate.

THE AMERICAN BOARD OF SURGERY, II

In answer to the widespread demand for an agency which will attempt to certify competent surgeons the American Board of Surgery has recently been organized. This Board is a member of the Advisory Board of Medical Specialties which includes all of the boards of certification for the different medical specialties which have been already organized. Since boards were in existence for the certification of practitioners of some of the surgical specialties such as ophthalmology, otolaryngology, obstetrics and gynecology, genito-urinary surgery and orthopedic surgery, it is expected that the American Board of Surgery will be responsible for the certification of general surgeons as well as those practicing in the remaining specialized subdivisions of surgery.

Acting upon the invitation of the American Surgical Association the following surgical societies cooperated in the creation of the American Board of Surgery: the American Surgical Association, the Surgical Section of the American Medical Association, the American College of Surgeons, the Southern Surgical Association, the Western Surgical Association, the Pacific Coast Surgical Association and the New England Surgical Society. The first three of these bodies which are national in scope have three representatives on the Board. All of the other societies have one representative each. The representatives of the cooperating societies are nominated by the society which they represent and upon approval of the Board shall become members of it. The term of membership on the Board will be six years. The following were chosen to represent the cooperating surgical societies:

Dr. Evarts A. Graham, St. Louis; Dr. Arthur W. Elting, Albany, N. Y.; Dr. Allen O. Whipple, New York; representing the American Surgical Association.

Dr. Donald Guthrie, Sayre, Pa.; Dr. Erwin R. Schmidt, Madison, Wis.; Dr. Harvey B. Stone, Baltimore; representing the American College of Surgeons.

Dr. Fred W. Rankin, Lexington, Ky.; Dr. Howard M. Clute, Boston; Dr. J. Stewart Rodman, Philadelphia; representing the Surgical Section of the American Medical Association.

Dr. Philemon E. Truesdale, Fall River, Mass.; representing the New England Surgical Society.

Dr. Thomas Orr, Kansas City, Kan.; representing the Western Surgical Association.

Dr. Robert Payne, Norfolk; representing the Southern Surgical Association.

Dr. Thomas Joyce, Portland, Oregon; representing the Pacific Coast Surgical Association.

PURPOSES

(a) To conduct examinations of satisfactory candidates who seek qualification by the Board.

(b) To issue certificates of qualifications to all those meeting the Board's requirements.

(c) To improve the opportunities for the training of the surgeon.

REQUIREMENTS

General Qualifications:

1. Moral and ethical standing in the profession satisfactory to the Board.

   The Board, believing that the practice of "fee splitting" is pernicious, leading as it does to a traffic in human life, will reserve the right to inquire into any candidate's practice with regard to this question.

2. Membership in the American Medical Association or, by courtesy, membership in such Canadian or other medical societies as are recognized for this purpose by the Council on Medical Education and Hospitals of the A. M. A. Except as here provided, membership in other societies shall not be required.

3. Those who have limited their activities to the practice of of surgery.

4. In exceptional instances the Board may, in its discretion, accept for examination candidates who have met all preliminary requirements and have been in practice from six to sixteen years but whose formal training does not comply with the full requirements to be exacted in the future.

The Board recognizes two groups of candidates eligible for certification.

(A) The Founders Group—those who have already amply demonstrated their fitness as trained specialists in surgery. Candidates from this group *on invitation of the Board* may make application and upon approval by the Board will be accepted without examination as qualified. This group will be selected from the following:

1. Those who from the time of the Board's organization, Jan. 9, 1937, hold the position of Professor or Associate Professor of Surgery in the approved medical schools of the United States or Canada.
2. Those who for fifteen years prior to the Board's organization have limited their practice to surgery and have met the general qualifications required.
3. Such members of the cooperating societies represented on the Board, in good standing Jan. 9, 1937, who may be invited to membership in this group.

(B) Qualified by Examinations—In addition to the general qualifications the requirements for this group shall be as follows:

(a) Professional Standing

1. Graduation from a medical school of the United States or Canada recognized by the Council on Medical Education and Hospitals of the A. M. A., or graduation from an approved foreign school.
2. Completion of an internship of not less than one year in a hospital approved by the same Council, or its equivalent in the opinion of the Board.

(b) Special Training

1. A further period of graduate work of not less than three years devoted to surgery taken in a recognized graduate school of medicine or in a hospital or under the sponsorship accredited by the American Board of Surgery for the training of surgeons.

This period of special training shall be of such character that the relation of the basic sciences of anatomy, physiology, pathology, bacteriology and biochemistry is emphasized. Knowledge of these sciences as applied to clinical surgery will be required in the examination.

Adequate operative experience in which the candidate has assumed the whole responsibility will be required.

An additional period of not less than two years of study and practice in surgery.

The above requirements, especially those referring to surgical training, are subject to change from time to time as the existing opportunities for training in this field of specialization may be broadened.

Examinations

The qualifying examination will be divided into Part I (written) and Part II (clinical, bedside and laboratory). In both of these parts a knowledge of the practical application of the sciences fundamental to surgery will be required as previously stated.

Part I

This may be given simultaneously in as many centers as the Board may determine suitable for the purpose. A candidate, to be eligible for Part I, must meet all requirements for Group B candidates. A card of admission to this part of the examination will be forwarded to the candidate from the Secretary's office, certifying that these requirements have been met, as well as due notice as to the time and place of examination.

The examination in Part I shall cover a one-day period. There shall be two sessions of three hours each. This written examination shall concern itself primarily with general surgical problems and in addition the application of the basic sciences of surgery to these problems.

Part II

In order to be eligible for Part II a candidate must have successfully passed Part I, in addition to having met the necessary preliminary requirements and having presented definite evidence of an adequate training in operative surgery to the Board.

This part of the examination shall be oral and practical and cover a two-day period.

It is probable that for the present this part of the examination can be held in one center or, at the most, two. Later, however, as the demand grows, it will be necessary, in all probability, for the Board to establish definite subsidiary board centers where this part may be held. At that time it will be necessary that the Board appoint subsidiary boards in these centers, consisting of those already qualified as in Group A, to conduct this examination.

Grades

A candidate must receive a passing average for each Part to be entitled to the Board's certificate. No candidate shall pass a Part who does not receive a grade of 60 per cent or over in each subject of such a part. An average grade of 75 per cent shall be considered as passing in each Part.

A candidate who fails in his examination in Part I shall have his papers reviewed by the Examination Committee.

Reexaminations

Candidates may be reexamined as often as they desire provided one year shall elapse between examinations, except that the Board may, for good and sufficient reason, deny a candidate the privilege of reexamination.

Candidates shall be required to pay the same fees for Parts I and II at each reexamination in these Parts.

Fees

The fee for Group A, Founders Group, shall be $25.

The fee for Group B shall be $75, payable as follows: $5 registration fee, which shall be returned if the candidate is not accepted for examination; $20 for Part I; and $50 for Part II.

This Board is a non-profit organization. All fees will be used, after a reasonable amount is set aside for necessary expenses in maintaining its office, conducting examinations, etc., to aid in improving existing opportunities for the training of the surgeon.

Certificate

A certificate attesting to a candidate's qualification in surgery after meeting the requirements will be issued by the Board, having been signed by its officers.

Revocation of Certificate

Any certificate issued by the Board shall be subject to revocation by the Board at any time in case it shall determine in its sole judgment, that a candidate, who has received a certificate, either was not properly qualified to receive it or has become disqualified since its receipt.

Proper forms for making application, and other information, will be furnished by the secretary

DR. J. STEWART RODMAN
225 S. Fifteenth Street
Philadelphia, Pa.

ELECTROSURGERY

In THE SOUTHERN SURGEON for August, 1937, on page 328, I note a paragraph in an article by Dr. Charles DeForest Lucas which interests me. The remainder of the paper is well worth reading, but this paragraph of only four lines is so at variance with the experience of an increasing number of reputable surgeons that it should not be accepted without fuller information.

Dr. Lucas states that the electric endotherm is exploited by its manufacturers and by "those seeking fame through innovation." He also goes on to say that the trained neoplastic surgeon would find the innovation of little use. It is unfortunate that this paragraph crept into Dr. Lucas' article, because if he will investigate the matter he will find that many of the surgeons using electrosurgery do so not for advertising purposes but because they are convinced that it has great advantages over the older methods. This, of course, does not apply in every operation, but it certainly does, according to my own experience, in the majority of incisional surgical procedures. It is hardly worth while to present here a bibliography of this subject, but the reader may be put on the right track by referring to an article by the writer, with the clinical report of 118 operations, "Electrosurgery" (*Annals of Surgery*, June, 1933.)

Since that time, this has been my method of choice as a first step in nearly all of my operative work, except that in which the skin incision, for some reason, must be made very slowly. This means that I begin nearly every operation by making the incision with the electro-thermic scalpel.*

To mention only a very few of the surgeons who have found electrosurgery of great value, I may include Dr. Harvey Cushing, Drs. Howard A. Kelly and Grant E. Ward, who have written a book on the subject, Dr. Beverly Chew Smith who has investigated the type of healing which occurs when the skin has been quickly divided by the electric scalpel.

Infection cannot be carried by the electric knife, and since coagulation is superseding the ligature, another carrier of organisms has been abolished. In the case of a rapid, sure incision, adhesion of the divided walls of skin occurs with amazing rapidity.

It will be noted that I have not referred to the mere electrical destruction of neoplasms; this being, to me, an obvious procedure.

HOWARD LILIENTHAL, M. D.

*This term, I think, was first used by Dr. George A. Wyeth, who perhaps more than any other man in America or elsewhere, has succeeded in developing this valuable operative advance.

GASTROSCOPY IN SURGERY

The Oslerian principle of routinely exploring every natural orifice of the body has, since the development of the flexible gastroscope in 1932 by Rudolf Schindler, been extended to the stomach. Such an inspection causes an ambulatory patient little more inconvenience than the passage of a stomach tube. When both physicians and laymen realize that such an examination can be done, and should be done when unexplained lack of appetite, anemia, loss of weight or digestive disturbance is observed in persons over 35, cancer of the stomach will be diagnosed much earlier than it usually is now. There is an urgent need for such early diagnosis for 30 to 40 per cent of all cancers observed occur in the stomach and 30,000 die annually in the United States from gastric carcinoma. It is not surprising that so many people believe that early cancer of the stomach is without symptoms and that by the time symptoms appear it is too late for successful surgical treatment.

All patients in whom gastric ulcer has been diagnosed should be gastroscoped immediately. Otherwise in cases of malignancy valuable time may be lost in waiting for response to medical therapy instead of giving the patient the benefit of prompt operation.

Gastroscopic differentiation of malignant from benign ulceration is made on their characteristic dissimilarity in appearance. The benign ulcer stands out as a bright yellowish punched out area in an orange-red field. It is usually round or elliptical. The edge is always sharp and does not merge into the adjacent mucosa. The floor is covered with a white or yellow-white adherent membrane and may show one or more dark red streaks or sometimes oozing blood.

In contrast, the edges of the carcinomatous ulcer are ragged and eroded with an infiltrated margin. The floor is irregular and may contain nodules and ridges in a dirty colored background which may vary from gray to red-brown or violet. Usually the malignant ulcer appears on an elevation. The differentiation of benign from malignant ulceration is emphasized by the multitude of colors which depend upon the circulation of the blood.

A benign epithelial tumor or the more common atrophic gastritis must be recognized as forerunners of carcinoma and, as potentially malignant, should be examined by gastroscopy twice a year. With the realization that the early diagnosis of gastric malignancy can be made, this condition will more often reach the surgeon at an operable stage.

In determining the operability of a lesion, we may be guided by gastroscopic inspection of its pathologic characteristics. The polypoid carcinoma and the non-infiltrating carcinomatous ulcer are al-

most always operable unless near the cardia. The infiltrating carcinomatous ulcer is always doubtful. The site of the lesion, stage, and direction of its infiltration must be considered in determining its operability. The diffusely infiltrating carcinomas are always inoperable.

Earlier diagnosis of gastric malignancy and the utilization by the surgeon of gastroscopic criteria in determining operability will inevitably improve surgical results. This will engender in both physicians and the public a more watchful, hopeful attitude, with a greater confidence in gastric surgery. The fight against gastric cancer will then take its place along with the brilliant and successful campaign against malignancy of the uterus and breast.

The development of gastric surgery has resulted in an increasing number of patients suffering with gastric symptoms after operations on the stomach. In meeting this recurrent postoperative distress there has been little recourse for the surgeon. Too often in the mind of the patient or the referring physician, the surgeon is undeservedly credited with a failure. This will not be true when it is realized that of all patients with gastric symptoms, 40 per cent show definite chronic changes in the gastric mucosa, which are comparable to the abnormalities found in other mucous membranes of the body. It is evident that symptoms of chronic gastritis which have no relation to any surgical procedure will occur in many postoperative patients. Gastroscopic examination, however, may reveal a true postoperative gastritis resulting from the failure of an artificial stoma to develop rhythmic adaptation. Gastrojejunal ulcer or a recurrent gastric ulcer may be discovered. Occasionally, a silk suture will cut through the mucosa and hang freely in the stomach cavity. This results in erosion with marked symptoms. Recurrence of a carcinoma may be seen as mucosal metastases or it may sometimes involve the stoma.

In postoperative conditions gastroscopy permits an evaluation of the pathologic changes. Such evaluation is based upon comparison of the normal with the abnormal. Gastroscopic findings in postoperative patients free from distress should be compared with findings in patients having unsatisfactory results. Systematic observation will materially aid in solving the problem of attaining physiologic function following anatomic alteration.

CRAWFORD F. BARNETT, M. D.

EXHIBITS

Two years ago in New Orleans with the friendly rivalry between Tulane and L. S. U. to give impetus to the preparation of exhibits and with the able assistance of the Marine Hospital, the scientific exhibits were the most elaborate ever shown before an Assembly of The Southeastern Surgical Congress. Except as regards numbers, they were quite comparable to those of the annual sessions of the American Medical Association. Last year, although the doctors of Charlotte worked themselves into a lather getting up an excellent program on two weeks' notice, exhibits were woefully lacking. The 1938 Assembly in Louisville, which city has now fully recovered from the great flood of last year, we expect at least to compete with the impressive exhibits provided by the New Orleans medical schools and the Marine Hospital—with the University of Louisville School of Medicine providing the nucleus.

Of course we are expecting the aid of other medical schools near and far, and we will welcome with enthusiasm exhibits by individual Fellows.

If you have something to show, write to Dr. Beasley. The space set aside is quite large but it is best to reserve your place early.

No feature of a medical convention is more educational than the exhibits: they are in themselves a postgraduate education. Papers presented can be studied and enjoyed later when they are published; not so the scientific exhibits—the gross specimens, the microscopic displays, the large charts, the colored drawings must be viewed during the Assembly. The exhibitors are not only willing but eager to explain their work to all who manifest an interest.

The technical exhibits are of hardly less educational value. These will include "the latest types of surgical instruments of all kinds, x-ray and diathermy paraphernalia, the newest books, the most recent improvements in pharmaceutical preparations and just about anything else in the world that will enable a man to give his patients the most up-to-date service." The exhibitors in this field are only too eager to show and talk about their products to any interested surgeon.

There will be a thirty minute intermission in the scientific program each morning and afternoon to allow everyone to study the exhibits, both professional and technical. THE SURGEON backs up the exhibits and everything is quite reliable. We heartily recommend them all.

BOOK REVIEWS

The Editors of THE SOUTHERN SURGEON *will at all times welcome new books in the field of surgery and will acknowledge their receipt in these pages. The Editors do not, however, agree to review all books that have been submitted without solicitation.*

A PHYSIOLOGICAL AND CLINICAL CONSIDERATION. THE THERAPEUTIC PROBLEM IN BOWEL OBSTRUCTION. By OWEN H. WANGENSTEEN, M. D., Ph. D., Professor of Surgery, University of Minnesota and Surgeon-in-Chief, University of Minnesota Hospital. 360 pages, with 90 illustrations. Price, $6. Springfield and Baltimore: Charles C Thomas, Publisher, 1937.

We have all read something of the newer methods of treating intestinal obstruction; we all know that "The duodenal tube has superseded the enema in the relief of intestinal distension"; most of us have had some experience in the application of the modern principles. Although we have known that Wangensteen started all this, his papers, as well as those of the many surgeons who have corroborated his work, have been scattered through various journals, and it has been difficult not to miss some detail of importance. It has been even more difficult to look up some point that has grown hazy in our memory.

Now the original essay that won the quinquennial Samuel D. Gross prize for 1935, together with the results of further clinical experience both of Dr. Wangensteen and his collaborators and of others, are presented in a book which is essentially an original work, and one of superlative importance. Every surgeon who enters the abdomen and every physician whose patient either spontaneously or postoperatively suffers from abdominal distention should study this book carefully and keep it where he can refer to it easily.

Dr. Wangensteen lays great emphasis on the importance of correct diagnosis before operating on an "acute abdomen." He insists that formulating the diagnosis is not a mere intellectual exercise for the surgeon: although exact diagnosis is often impossible, the more exact the diagnosis the better chance the patient has. His discussion of the differential diagnosis is in many respects radically different from the traditional one that has been copied from one textbook into another. It is so up to date that it includes dissecting aneurysm.

The author lays great stress on the value of x-ray (it goes without saying that he does not use barium) in determining the site of obstruction and in following the course of the patient. Of course the significance of blood chemistry is given in some detail, and the importance of supplying chlorides when the blood chlorides are down is emphasized. The book should serve to remind undergraduates that biochemistry and physiology are not disagreeable preludes to clinical surgery but fundamental parts of it.

Dr. Wangensteen's studies, both experimental and clinical, of intestinal obstruction probably constitute the greatest advance in surgery during the past ten years. If for no other reason, the reviewer advises the purchase of this book as an investment for he believes that within a century a first edition will rank in value with a first edition of Beaumont. However, lest he be thought to be carried away with enthusiasm or perhaps not to have read it carefully, he must find a flaw: Dr. Wangensteen's skill in dissolving the continuity of tissue is so mirrored in his literary style that he has not missed an opportunity to split an infinitive.

DISEASES OF THE BLOOD AND ATLAS OF HEMATOLOGY WITH CLINICAL AND HEMATOLOGIC DESCRIPTIONS OF THE BLOOD DISEASES INCLUDING A SECTION ON TECHNIC AND TERMINOLOGY. By ROY R. KRACKE, M. D., Professor of Bacteriology, Pathology and Laboratory Diagnosis, Emory University School of Medicine; Pathologist to the Emory University Hospital; Consultant in Hematology to the Grady Hospital and Egleston Hospital for Children, Atlanta, Ga.; formerly, Director of the Hematological Registry, American Society of Clinical Pathologists, and HORTENSE ELTON GARVER, M. S., Instructor in Laboratory Diagnosis, Emory University School of Medicine. 532 pages, with 44 color plates and 17 other illustrations. Price, $15. Philadelphia, London and Montreal: J. B. Lippincott Company, 1937.

Not so long ago diseases of the blood were considered uniformly fatal and the average physician felt little interest in them. Kaznelson's brilliant success in curing purpura hemorrhagica by means of splenectomy turned the surgeon's hopeful interest again to this subject. Following this the epoch-making discoveries as to pernicious anemia, and the drama of agranulocytosis made hematology one of the most fascinating fields of medicine, and one that was well harrowed. However, it seemed that nearly every researcher wished to coin his own terms, and there was grave doubt that two specialists in this line though living in the same city could understand each other's ideas. It was therefore necessary for some one of authority to publish in English a book that could bring order out of chaos and end the confusion. No more fitting person could have undertaken the task than Roy R. Kracke who has so often been honored by his peers.

Dr. Kracke has called to his aid in preparing the text several other experts. This text represents the seasoned evaluation of the work of recent years. It is clearly written and of great value not only to him who wishes to have an up-to-date summary of work that has been completed in order to extend his research but also to the clinician. It is of course indispensable to the student and to the laboratory technician.

Dr. Kracke has taken full heed of the old maxim that one good picture is worth many pages of text: Mrs. Garver's forty-four drawings which are reproduced in full colors (the Editor shudders to think what their reproduction must have cost!) make the volume invaluable. It is probable that the only rival atlas is that of Naegeli published in Germany some years ago.

While surgeons cannot be expected to train themselves as expert hematologists the text will provide them with useful information; they should have access to the text when the possibility of a blood dyscrasia enters the differential diagnosis. They should require that the laboratory of the hospital they work in have this book on hand for constant reference.

In passing, Dr. Mays' paper in the present issue emphasizes the practical need of a knowledge of hematology.

THE ART OF MINISTERING TO THE SICK. By RICHARD C. CABOT, M. D., and RUSSELL L. DICKS, B. D. 378 pages. Price, $3. New York: The Macmillan Company.

One of the greatest advances in the practice of medicine during the past decade (excepting perhaps the introduction of para-amino-benzene-sulfomide) has been the return to the realization that the patient is a personality—that in the care of many of them, the handling of their fears and worries is hardly

less important than the surgical correction of their ailments. The art of ministering to the sick requires no less skill and much more time than the scientific treatment of them, but this art is no less demanded of the truly successful doctor.

Man moreover is innately a religious animal. The physician who can answer spiritual needs of his patients is, to our shame be it said, too rare. The clergyman should be in a better position to care for these—such duties are more essentially in his line, and he has more time for them. Again however, some preachers are lacking in tact, tending, if not unduly to alarm the patient, to exhaust him; and some do not realize that they should consult with the physician in charge, and show due consideration for the convenience of the nurses.

The conscientious doctor who wants his patient to have the best and the earnest clergyman who wants to do all possible for his sick parishioners and perhaps other unfortunates, will both derive great benefit from this great book, "The Art of Ministering to the Sick."

A TEXTBOOK OF MEDICINE. By AMERICAN AUTHORS. Ed. by RUSSELL L. CECIL, A. B., M. D., Sc. D., Professor of Clinical Medicine, Cornell University Medical College; Associate Attending Physician, New York Hospital, New York City. Associate Editor for Diseases of the Nervous System, FOSTER KENNEDY, M. D., F. R. S. E., Professor of Neurology, Cornell University Medical College; Director, Department of Neurology, Bellevue Hospital, New York City. Fourth edition, entirely revised and reset. 1,614 pages, with 42 illustrations. Price, $9. Philadelphia and London: W. B. Saunders Company, 1937.

No up to date surgeon would think of driving to the hospital in a car that made its first appearance more than ten years ago and yet many, it is to be feared, keep in their offices the textbook of medicine that they used in their undergraduate days as their only work of reference in non-surgical matters. The surgeon of course can't be expected (nor can anyone else) to keep fully abreast of every advance in general medicine and yet unless he is closely allied with competent internists he ought to have an up to date book in this field for ready reference. The new edition of Cecil, the fourth in ten years, will meet his needs admirably.

Some 160 authors have contributed to its success. It is interesting to note that the editor and the publishers have established a retiring age for contributors "though such a course necessitated the loss of many excellent articles, it had the advantage of introducing new blood into the textbook." It is extraordinary how much authentic material is compressed into such a short space. It should indeed prove invaluable to every M. D. as a first work of reference.

Correction. In writing of Warbasse and Smyth's Surgical Treatment in the October SURGEON it was stated that there were several omissions: Jacobaeus' method of severing pleural adhesions will be found in Vol. 2, p. 398; Beck's cardiorrhaphy for angina is mentioned in Vol. 2, p. 412, and the injection treatment of hemorrhoids is discussed in Vol. 3, pp. 44 and 45. Two other subjects that were said to have been omitted can be located if one knows where to look for them.

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Book reviews are grouped together and are indexed under that heading in alphabetical order.

MISSISSIPPI VALLEY MEDICAL SOCIETY AWARD

The Mississippi Valley Medical Society offers a cash prize of $100.00, a gold medal and a certificate of award for the best unpublished essay on a subject of interest and practical value to the general practitioner of medicine. Entrants must be ethical licensed physicians, residents of the United States and graduates of approved medical schools. The winner will be invited to present his contribution before the next annual meeting of the Mississippi Valley Medical Society (Sept. 28, 29, 30, 1938), the Society reserving the exclusive right to first publish the essay in its official publication—the Radiologic Review and Mississippi Valley Medical Journal. All contributions shall not exceed 5,000 words, be typewritten in English in manuscript form, submitted in five copies, and must be received not later than May 15, 1938. Further details may be secured from

HAROLD SWANBERG, M. D., Secretary,
Mississippi Valley Medical Society,
209-224 W. C. U. Building, Quincy, Ill.

Statement of the ownership, management, circulation, etc., required by the Acts of Congress of August 24, 1912, and March 3, 1933, of The Southern Surgeon, published bi-monthly at Atlanta, Georgia, for October, 1937.

State of Georgia, County of Fulton, *ss.*

Before me, a Notary Public in and for the State and county aforesaid, personally appeared B. T. Beasley, who, having been duly sworn according to law, deposes and says that he is the Business Manager of THE SOUTHERN SURGEON, and that the following is, to the best of his knowledge and belief, a true statement of the ownership, management, etc., of the aforesaid publication for the date shown in the above caption, required by the Act of August 24, 1912, as amended by the Act of March 3, 1933, embodied in section 537, Postal Laws and Regulations, to wit:

1. That the names and addresses of the publisher, editor, managing editor, and business manager are:

Publisher, The Southern Surgeon Publishing Company, Atlanta, Ga.; Editor, Dr. L. Minor Blackford, Atlanta, Ga.; Managing Editor, Dr. B. T. Beasley, Atlanta, Ga.; Business Manager, Dr. B. T. Beasley, Atlanta, Ga.

2. That the owner is: The Southeastern Surgical Congress, Atlanta, Ga., a non-profit organization.

3. That the known bondholders are: None.

B. T. BEASLEY, Managing Editor.

Sworn to and subscribed before me this the 1st day of October, 1937.

MARGARET H. CHAPMAN, Notary Public.
(My commission expires April, 1939.)

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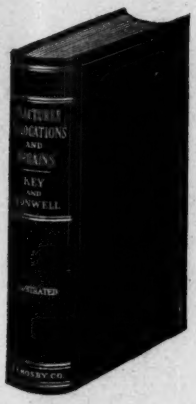

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